UNITED STATES BANKRUPTCY COURT
FOR THE WESTERN DISTRICT OF NORTH CAROLINA
CHARLOTTE DIVISION

IN RE:	)	
	)	
GARLOCK SEALING TECHNOLOGIES	)	
LLC, et al,	)	No. 10-BK-31607
	)	
Debtors.	)	VOLUME IV-B
	)	AFTERNOON SESSION

TRANSCRIPT OF ESTIMATION TRIAL
BEFORE THE HONORABLE GEORGE R. HODGES
UNITED STATES BANKRUPTCY JUDGE
JULY 25, 2013

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I N D E X

THURSDAY AFTERNOON, JULY 25, 2013

(Called to order at 1:48 p.m.)

THE COURT: I'll warn you all that I made a mental note of Mr. Finch's birthday; and if we are here on August 9, we will conclude by singing happy birthday to him. And I'll offer that as some incentive to move this thing along.

Okay. Mr. Schachter.

MR. SCHACHTER: I'll be as expeditious as possible, Your Honor.

We call Dr. David Weill to the stand, please.

DAVID WEILL,

being first duly sworn, was examined and testified as follows:

DIRECT EXAMINATION

BY MR. SCHACHTER:

Q. Dr. Weill, would you introduce yourself to the court, please.

A. I'm David Weill.

Q. And what's your occupation, sir?

A. I'm a physician at Stanford.

Q. Sir, you followed a witness who has described the culmination of an extensive process: Information gathered over months and, I guess, years about the current claimants in an effort to understand them, and likely future claimants. This information has been analyzed and evaluated. And information has been grouped about that.

You're aware of that process?
A. I am.
Q. And have you reviewed the results of Mr. Henshaw's evaluation of the exposure, the annual cumulative exposure of the various groups that he described only briefly this morning?
A. I have.
Q. Will you be able to give us the medical understanding of that exposure?
A. Yes, I will.
Q. And the key issue that we're going to focus on, there are two issues, is whether gasket exposure in and of itself is sufficient to be a cause of mesothelioma; and then the larger issue that we contend is raised by the law, the issue of specific causation, whether, in the context of the total lifetime exposure of likely claimants, their gasket exposure from asbestos is a substantial contributing factor.
You've understood that to be your role?
A. Yes.
Q. Well, let's find out a little bit about why you're entitled to -- or should be able to talk about that. Tell us about your certifications. What are you certified in?
A. I'm boarded in pulmonary medicine and critical care medicine.
Q. And your training you got at what schools?

A. University of Colorado where I did my pulmonary and critical care training in addition to my lung transplant training, and I received my undergraduate and medical degree from Tulane University.
Q. Currently you are at Stanford University Medical Center. Is that the right name for the institution?
A. Yes.
Q. And what do you do there, sir?
A. I direct the Center for Advanced Lung Disease and also am the medical director of the lung and heart/lung transplant program. I serve as professor of medicine in the division of pulmonary critical care medicine.
Q. And does that involve teaching responsibilities?
A. Yes, it does.
Q. Do you also maintain an active clinical practice, sir?
A. I do.
Q. Tell us just a little about that.
A. Our center is involved, as the name implies, in a variety of lung diseases. We provide both novel medical therapy for those diseases and also provide an option for surgical therapy such as lung transplantation for certain kinds of lung disease.
Q. Are you a doctor who has hands on regular contact with patients?
A. Yes, almost every day.

Q. Recently there was an unfortunate tragedy up in San Francisco, a plane crash.
A. Yes.
Q. Were you involved in the care of any of the people that were injured in that?
A. In the critical care capacity, yes.
Q. And in terms of your lung transplantation work, what kind of involvement do you have in that process, sir?
A. I direct the program and then the clinical involvement is primarily with regards to deciding who is a candidate for lung transplantation and who is not. I also pick the lung donors for those patients. I then also -- and when I say "I," I mean my team as well as myself, takes care of the patients beginning in the intensive care unit and all the way out through the rest of their life.
Q. Can you explain to the court what your background is in issues of asbestos-related lung disease and mesothelioma.
A. Where I went to medical school was interested in occupational lung diseases of all sorts and so became interested at that time. So this would have been in the late '80s. And throughout my career, I have taken care of patients with occupational lung diseases in addition to a variety of other lung problems, and have had the opportunity to participate in the writing of book chapters, for instance, and done some original research.

Q. And you've written a book chapter in a book that was recently published by the Oxford University Press on "Asbestos and Its Diseases." Are you a chapter contributor in that book?
A. Yes, I am.
Q. And there is another book, "Hunter's Diseases of Occupations, 2011." Are you a chapter contributor to that book on an issue related to asbestos disease?
A. Yes, I am.
Q. Sir, in addition to your -- have you personally been involved in caring for and treating mesothelioma patients?
A. Yes, I have.
Q. Can you give us just some range? I know it's a rare disease and it's rare for doctors to see it, but how many --
A. Probably over the course of my career, which would include my training as well, I've cared for between 40 and 50 of these sorts of patients.
Q. Now, in addition to that, there's sort of a family background in asbestos research and medicine. We're going to see some names in the asbestos history that we're going to talk about. Can you explain your connection there.
A. My father led an occupational lung disease group at Tulane University that did a fair amount of the original research on asbestos-related diseases as well as other occupational lung diseases.

Q. So when we talk about some of the luminaries of the asbestos research, Dr. Selikoff and Sir Richard Doll and some of these other people, you actually have met some of these people.
A. I have.
Q. Sir, in brief summary, can you just tell us what your opinions are in summary form.
A. Yeah, I'm going to offer four basic opinions today.
One is going to be a discussion and the notion that lung defenses prevent very small asbestos exposures from harming us, and I'll go through the reasons for that.
I'm also going to offer the opinion that exposure to gaskets and packing do not elevate the risk of mesothelioma, but instead, asbestos-related mesothelioma cases are caused by amphibole type of asbestos exposure.
And then given the assumptions that are out there and the work that's been done by Mr. Henshaw and others, I'm going to opine that Garlock exposure was not a substantial cause in the groups identified by his study.
Q. And thank you.
MR. SCHACHTER: I didn't offer the witness as an expert, but I here -- before getting the basis of his opinions and I apologize to the court. I hereby offer Dr. Weill as an expert in asbestos disease and pulmonary medicine.
MR. GEORGE: No objection, Your Honor.

THE COURT: He will be admitted as such.
Q. Sir, I'd like to start, if I may, by having you explain to us the anatomy and the nature of the human body's reaction to asbestos disease. Are you prepared to do that, sir?
A. I am.
Q. Would it help if you came down here to go through your slides so you can explain them to the court?
A. Sure, I think so.
(Witness stepped down from the witness stand.)
Q. Dr. Weill, can you explain the lung's or the human body's defenses to mesothelioma and where the disease starts and the orientation of it, please.
A. Sure. Where we're going to talk about with regards to mesothelioma is really the lining of the lung called the pleura. The pleura is a surface that encases the lung and it's made up of two parts. One is called the visceral pleura which is connected physically to the lung. And one is called the parietal pleura which adheres more closely outside of the lung to the chest wall.
Q. And if we -- then what are the body's defenses?
A. So basically, if you break down the body's defense mechanisms, you talk about two major components.
One is the physical defense systems that are in place. Those primarily comprise of physical barriers to things that we inhale from getting down into the deep parts of the lung.

And the second broad category of defense systems is the cellular defense systems. So after the physical defense and only if the physical defense systems are overwhelmed does the cellular defense mechanisms kick into place.

So first -- then a discussion of the upper respiratory tract defenses.

What you've got is, first, nose and mouth. So you've got nasal hairs, the tongue itself, turbinates which are bones in the nasal pharynx. And all of these structures create a situation where hopefully fibers or anything that we inhale will impact on those structures before they get down deep into the lung.

There's also, importantly, vocal cords which sit further down which provide us our cough reflex. So we all know that when something hits our vocal cords it causes us to cough that substance out.

Q. Let me hand you the clicker as you go through it, the next slide. That way you can control the speed.

A. Sure. And so as we move through the physical defense systems, we have the trachea, the main airway, dividing into two bronchi, left and right mainstem bronchi. And all along those structures we've got defense mechanisms in place that help protect us.

So what you've got is you go further deeper into the lungs. The passages narrow and the lung divides 23 times.

And so that in order for a fiber or particle to get deep down into the lung, the fiber or particle has to navigate these divisions.

Q. Before we -- okay. And you call that what? Mucociliary escalator. What is that, sir?

A. So the mucociliary escalator sits on the large airways primarily. And what the purpose is, as the name implies, is that you've got a cilia layer that has on top of it mucous. And so what the cilia does -- and this is oriented upright as if the trachea wall is here, for instance. The head is up here. Feet are down here.

What you've got is the cilia beating this way. So they beat from the lower portion of the body, or south, northward toward the mouth and the nose. And what happens is is that when you inhale something, the mucous layer traps the fibers and particle along that mucous layer and the cilia then beat northward to either cough, sneeze or swallow those fibers and particles out.

Q. So what direction do the fibers move?

A. From the lower part of the airway toward the mouth and the nose and throat.

Q. Okay. The next slide, and I guess that's a cilia and mucous, and the fiber travels upward.

A. Yes.

Q. Okay. So the fibers go up on the mucociliary

escalator.

Now, our anatomy lesson I'd like to interrupt by pointing out a statement.

Do you know this man, Irving Selikoff?

A. Yes.

Q. And the court has heard this before. He has written that "It is fortunate that the greatest part of the asbestos in construction materials has been in products in which the asbestos is locked in - that is, it is bound with cement or plastics or other binder so that there is no release, certainly no significant release, of asbestos fiber in either working areas or general air."

And is it clear that science recognizes the difference between encapsulated products and friable products?

A. Yes, and has for some time.

Q. What is that difference? I shouldn't be explaining it, you should.

A. The real difference from a pulmonologist's perspective is in respirability: The ability of those fibers to get down into the deep portions of the lung and cause trouble.

So I think what Dr. Selikoff -- I think what Dr. Selikoff was trying to point out here is that when you're considering whether or not something is respirable or not, one needs to understand the fiber characteristics as well as you can. And so that's why he's making a difference between encapsulated

and non-encapsulated fibers.
Q. So one difference that he made in the '70s was that there is a very low release of fibers. But do we know from subsequent research anything about the nature of those fibers as it affects their ability to penetrate into the deep zones of the lung?
A. There's been additional work on that as well.
So I think that this photomicrograph shows the morphology of an encapsulated asbestos fiber. And what you see here is an asbestos fiber that's got associated with it an encapsulating material. And the point of showing this is that the encapsulation doesn't have to necessarily be complete around the fiber in order for that encapsulation to affect the respirability of that fiber. And the reason it does so is because the aerodynamic properties of even a partially encapsulated fiber are altered when there's encapsulation around it.
And so a fiber typically would go down the airway in a fairly linear fashion and wobble a little bit.
Q. Yeah.
A. When it's got encapsulation around it, whether complete or partial, it wobbles more because it loses it's arrow dynamic properties just as if you were riding a bike and instead of having one of those cool fancy helmets, you have one big bulky helmet going down the road.

And so what you've got then is a wobbling of that encapsulated fiber and that limits its ability to be respired deep into the lung.
Q. So as it has to -- what did we say, there were 23 branches that it had to get through?
A. Right.
Q. And every time -- is that sort of like making a turn?
A. Yes.
Q. And it's trying to turn with that encapsulating on it and it's more likely to get stuck in the top, is that your point?
A. That's exactly right.
Q. All right. But obviously, even -- are you saying that every fiber from an encapsulated product is caught in the upper respiratory system?
A. No, not necessarily every fiber.
Q. And then if it makes it past the upper respiratory system, are there any more defenses that protect the body?
A. Yeah. There's a cellular type defense mechanism that kicks in when a fiber or particle has defeated the physical defense system.
Q. Okay.
A. And so this is work that happens deep in the lung and therefore I've labeled it on the slide deep defenses.
Q. And are there several of those deep defenses?
A. Yes. And the major player in the deep defenses of the

lung is the macrophage.
Q. Okay. And the macrophage, what is that? How does that work?
A. The macrophage is a cell that's sort of a Pac-Man like structure that can migrate toward fibers and particles, engulf them, and then use enzymes to break down the fiber or particle.
Q. Okay. In this diagram where are we in the body?
A. So this is -- this is then, in this case, the left mainstem bronchus dividing off into more minor bronchi, and then you've got alveolar structures, these little sack like structures, that sit in the periphery of the lung.
Q. And where is the pleura in this diagram?
A. The pleura is out here on the edge of the lung. It's the lining of the lung.
Q. Okay. So if we are looking here, the individual alveoli, what are they again?
A. So the alveoli is actually the working part of the lung. It's where carbon dioxide is expelled and oxygen is taken up. And so it's way out in the periphery of the lung. And in each alveoli there is at least one macrophage that's involved in eating fibers and particles that come into contact with it.
Q. And how does that work?
A. It works in really two major ways. One is macrophage migration. So a macrophage can sit in the alveolar structure,

as it's doing on this slide, and it can be activated by a foreign invader. And so when that happens, whether it's a particle or fiber like I've shown here, the macrophage can move to these particles and fibers and work on trying to digest it.

Q. And how do they do that, sir?

A. They have certain, what's called chemotactic factors which allow them to move.

Q. Yes, sir.

A. And then once they move to the fiber or particle, they engulf it and then release enzymes which break down the fiber or particle.

Q. And can they do that for all kinds of inhaled particles?

A. They try to do that for all kinds of inhaled particles, but depending on the fiber type, do it less well or well.

Q. We have heard from other witnesses that there are several different distinct minerals that are called asbestos. What are the most important ones for our analysis here to understand?

A. So the two major groups of asbestos type fibers are the amphiboles and the serpentines. The amphiboles have six, essentially six variants to it and the serpentines have one, namely chrysotile.

Q. Okay. And in dealing with these asbestos minerals of different types, does the body react differently to them or is

it more or less effective in its defenses against them?
A. It does. The body reacts similarly, but the end result is different.
Q. How is that, sir?
A. And so I approach the problems that asbestos inhalation can present from a lung perspective since I'm a pulmonologist.
Q. Sure.
A. So the understanding of what happens when a certain type of fiber is inhaled is very important to me and my understanding of these diseases. So for instance, if the lung macrophage encounters an amphibole like fiber, it's very difficult for that macrophage to defeat, digest, eliminate that amphibole fiber. And that's why amphiboles are more likely to cause disease than are chrysotile because chrysotile fibers are more easily able to be eliminated or digested by the macrophage system.
Q. Okay. Now, we've heard that fibers of chrysotile can reach the pleura. Do fibers of amosite reach the pleura as well?
A. Yes.
Q. Okay. And is there a difference of how the pleura deals with the fibers when they get to the pleura?
A. I think there is.
Q. How -- would you explain that us to, sir.
A. I think it's the ability of the fiber types and the

differences in the fiber types in their ability to be eliminated through the lymphatic channels that sit on the pleura's surface.

Q. Do you have some slides that illustrate that, sir?

A. I do.

Q. What is this?

A. So just for orientation purposes, the major part of the lung is out here to the left of the slide. This is an alveolar structure that's right next to the visceral pleura which is one of the layers of the pleura. What you've got in the middle is lymphatic channels running through the pleural surface and then you see a parietal pleura which is that part of the pleura which is next to the chest wall.

And the lymphatic system is in charge of eliminating things that come through the alveolar structures and out through these lymphatic channels. And you can think of them as a canal system to get rid of waste from the lung.

Q. And that canal system, does it work differently with long or short fibers?

A. It does.

Q. And has there been literature that has documented this in the peer reviewed medical literature?

A. There has been.

Q. How does that work?

A. I think the most eloquent description of how this works

was in an article by Ken Donaldson in 2010. And as the schematic shows, you've again got alveolar structures here. You've got the lymphatic channels. And you've got these smaller particles and fibers which are going out through this canal system easily. So in other words, they pass from the alveolar structures through the opening in the lymphatic system or the stoma and then out through the lymphatic channel to be eliminated from the lung.

Q. What if they are long fibers that haven't been digested by the macrophages?

A. Different situation. And so what you see in this schematic is lung fibers that are trying to be eliminated through the stoma in the lymphatic system. And instead of being eliminated, they essentially can't pass through the stoma because they're too big. And when that happens, an inflammatory cascade is set up such that reactions in the lung that can be deleterious occur on the pleural surface simply because these amphibole longer fibers can't be eliminated effectively.

Q. We heard something about black spots from Dr. Sporn.

A. Yes.

Q. And we saw a picture of black spots. Where are the black spots, where do they occur?

A. Well, the black spots, then, would occur right where this inflammatory process occurs on the pleural surface.

Q. Thank you, sir. So we've gone through the defense mechanisms and that's one explanation for why there's a difference between the longer amphibole fibers and the shorter chrysotile fibers in disease causation; is that correct?
A. Yes.
Q. And of course, the chemical -- we've already heard that there are chemical differences between the fibers. And from a medical standpoint, being there at Stanford, have they yet figured out all the genetic sequencing by which mesothelioma occurs?
A. No.
Q. Is it even figured out that it's definitely one tumor type or do we know a lot about that?
A. No, that has not been all worked out: How many genetic mutations have to occur, which ones are the exact ones, and what the impetus for those are.
Q. I think we've heard from witnesses for Garlock and I think that you'll probably hear even from the witnesses for the committee that epidemiology is the key to understanding disease causation for asbestos-related diseases, especially mesothelioma. Do you agree with that, sir?
A. I do.
Q. We've also heard a lot about different documents and what different scientists or a have said at different times about mesothelioma. To put that in context, would you be able to

track for us the history of the development of knowledge so far about mesothelioma and its causation?
A. Yes, I can.
Q. Where would you like to begin?
A. I think what you can do is start at the pre-1960s era.
Q. Yes.
A. And then we can work by decade up until the current time.
Q. Okay. Before 1960 was mesothelioma widely reported on or?
A. No. Most of what you saw in the medical literature before the 1960s involved very high dose exposures and primarily a focus on asbestosis.
Q. All right. And what happened in 1960 that changed the focus a little?
A. I think it was with Dr. Wagner's case series from South Africa where he saw a large number of pleural mesotheliomas clustering in the South African crocidolite mining population that he studied.
Q. What did he find, sir?
A. He found a total of 33 mesothelioma cases associated with, as the quote says, exposure to crocidolite asbestos, which is an amphibole type asbestos
Q. Okay. And his statement at that time was that the probable exposure to crocidolite exposure asbestos was the cause, right?

A. That's right.
Q. And he said that it was what, rarely seen elsewhere?
A. That's right.
Q. Now, this was a case series. Did -- with that case series, did that definitively establish the issue of asbestos relation to mesothelioma or was these words probable important in our understanding of things?
A. I think -- I think he was well advised to use the word probable because a case series is not proof of causation. But instead, I think it should be an impetus to do further search, which happened over the ensuing decades.
Q. Okay. And what was one of the next significant facts that occurred in additional research?
A. Not long after Dr. Wagner's discovery, Dr. Selikoff continued to be interested in this area and called for more research. And so he began in the mid '60s to wonder whether or not there was something else going on to cause mesothelioma other than crocidolite.
Q. Okay. Did they find in the early '60s additional cases of mesothelioma associated with crocidolite use in other countries?
A. Yes.
Q. And what other countries?
A. In the UK. In Great Britain.
Q. All right. And in 1965, what was the conclusion of the

scientific community about it?
A. That there was likely a causal relationship between crocidolite and mesothelioma.
Q. All right. Was there further concern?
A. I think the appropriate question then became -- that Dr. Selikoff then spent the next decade studying is whether or not crocidolite was the only type of fiber associated with an elevated risk of mesothelioma.
Q. All right. And as a result, there was a big conference, the Selikoff conference in the '60s.
A. Yes.
Q. Did they authorize or suggest that other places where asbestos was used start doing more research into this issue?
A. Yes.
Q. And did research indeed occur?
A. It did.
Q. Okay. Dr. Selikoff himself by 1972 had discovered what about amosite?
A. There was an interest in this particular amosite insulation manufacturing factory where they found mesothelioma in an amosite-exposed cohort that had nothing to do with crocidolite. So then the notion became not only is crocidolite a likely cause of it, but amosite probably is as well.
Q. All right. And were they also beginning a very famous

cohort of insulators at that time?
A. They were.
Q. Tell us about that.
A. So the Selikoff insulators, as they're called, ultimately resulted in 17,800 insulators being followed over a period of time, and a number of important discoveries came out of that insulator cohort.
Q. Okay. Ultimately, what was the death rate in that insulator cohort from mesothelioma alone, sir?
A. Ten percent of the insulator cohort died of mesothelioma.
Q. Did those insulators have high dose exposure to amosite-containing products?
A. They did.
Q. But there was also studies at plants where only amosite was used over time.
A. Yes.
Q. And those studies found what about mesothelioma?
A. That it also seemed to be related to the amosite exposure.
Q. All right. We've heard a little about Quebec. Was research begun in Quebec after the conference in the 1960s?
A. So about several years later, then, the Quebec cohort started.
Q. Okay.
A. And the publications began in the early 1970s. And this

followed a very large number of Quebec chrysotile miners and millers and found a difference in the number of different mesothelioma deaths. And so the question then became are there differences in fiber type potency as it relates to mesothelioma?

Q. But we've heard that the Quebec asbestos mining association funded some of that early research.

A. Yes.

Q. Was the fact that excess deaths were occurring in Quebec widely publicized in the 1970s and the early '70s by this McDonald group?

A. Yes. They published their finding.

Q. Okay. And then did they continue to follow their cohort over time for many decades?

A. Many decades.

Q. Would it take several decades before more definitive statements could be made about what the cause was?

A. Absolutely.

Q. In the interim was there also another kind of research going on into the nature of mesothelioma?

A. There was in the 1970s quite a bit of interest in doing animal experimentation on the relationship between asbestos exposure and mesothelioma.

Q. And what -- how did that work? What did they do?

A. Well, although animal studies are not directly analogous

to human experience because of differences en route of the administration of the asbestos or differences in dose or, in fact, just differences in the way animals respond as opposed to humans, some useful findings began to be elucidated at that time. And I put one on the slide here by Dr. Wagner looking at the inhalational animal experience by fiber type.

Q. Okay. In your report in this case, sir, have you dealt with more of the animal studies?

A. Yes.

Q. And if we go to that report, is there more detailed discussion that we really don't have time to go through?

A. There is.

MR. SCHACHTER: Your Honor, the report is in evidence but we may be referring to it a few times. May I give a copy to the court?

THE COURT: Yes.

(The document was tendered to the court.)

Q. Could you explain to the judge what the Wagner studies were finding.

A. The Wagner studies were interesting in that they found that -- not surprisingly, that animals produce mesothelioma in reaction to amphibole type inhalations. They also found, though, that there was a difference in the ability of even chrysotile inhalations in animals to cause tumors. And in fact, it looked as though from this study that only the

Canadian type of chrysotile was causing rat mesotheliomas as opposed to the Rhodesian, called at that time Rhodesia, no tremolite type of chrysotile.

Q. And in those studies, it took them massive exposures to rats in confined cages for a long time to even induce those, right?

A. That's right.

Q. And for these experiments, they used rat strains that were -- had a more suseptibility to get mesothelioma?

A. Yes.

Q. Okay. Well, let's continue with the epidemiology as it was developing. There's a study, a famous study by Acheson. Can you tell us about that, sir.

A. So the Acheson study published in 1982 looked at two gas mask manufacturers, gas masks being made in the UK at this time, in two different locations: One was in Leyland and one was in Blackburn.

Q. And did they make the same product?

A. No. The one in Leyland made crocidolite-containing gas masks that were used for the military, meeting military specifications.

Q. Right.

A. And the Blackburn factory made gas masks that contained chrysotile.

Q. And although both plants were making gas masks, one was

crocidolite and one was chrysotile, did they have equal or different rates of mesothelioma?

A. No, they didn't. The Leyland plant/factory produced mesothelioma, and I think there was a total of five cases. And the Blackburn experience was that there was one mesothelioma in that factory, but that was attributed by the authors to previous work with crocidolite the individual had done.

Q. Okay. Just so it's clear, how did the authors explain the difference?

A. They said that the probable explanation for the differences between Leyland and Blackburn lies in the different nature of the exposures in the two factories. The most obvious difference was that at Leyland crocidolite was the principal type of fiber.

Q. By 1983 when a number of the risk assessments we've heard about were starting to be written, did they have as much information as we have now, 30 years later, about asbestos and mesothelioma?

A. No, not at all.

And what I did on this slide, and this will be the first of a few chronology type of slides I use, is I filled in the study setting, the fiber type and the percentage of mesothelioma deaths by fiber type.

And so what you've seen in this first effort in 1983 to

fill in a matrix like this is that the large percentage of mesothelioma deaths were seen in the amphibole-exposed populations. And as you got further down toward a mixed exposure or a chrysotile exposure, the number of mesothelioma deaths either diminished or went away all together.

Q. And to continue, by 1987 had there been some more studies that had been done?

A. Yes, and I filled those in. Some were by Dr. McDonald, others by Dr. Berry. And they were in different exposure settings, but the message is the same and the sort of gradient that I indicated from the top where amphibole exposures caused a lot of mesothelioma deaths diminished then down to zero when you were looking at the chrysotile cohort.

Q. By 1989 had there been further development in the literature?

A. There had been. This is an IARC publication where Dr. Sir Richard Doll commented about what would -- what he felt was known at that time about fiber potency differences.

Q. Okay. And what did he say?

A. He said that "there is a difference between the effects of chrysotile and amphiboles, which is so great in relation to mesothelioma that it is possible to argue that chrysotile does not cause mesothelioma at all and that the relatively few cases that have occurred in men occupationally exposed to chrysotile have been due to the presence of an unintended

contamination with minute amounts of tremolite."
Q. Who was Sir Richard Doll?
A. He was a preimminent epidemiologist in his era making a lot of important contributions.
Q. In terms of the relationship between smoking and lung cancer, who's the foremost researcher on that?
A. Very much Dr. Doll.
Q. Is there any way you can accuse him of being a shell for industry as these lawyers have tried to --
A. No, I don't think so.
Q. Okay. That we've heard about doubt science and other things. Is that the kind of thing he'd engage in?
A. No, I don't think so.
Q. In the '90s did we have more information gathering as these studies start coming in?
A. Some in the 1990s were new studies that I highlighted in yellow down here. And then some were simply filling in more established cohorts with mesothelioma death rates. As I mentioned, the Selikoff insulators, between 9 and 10 percent; and the cigarette -- crocidolite cigarette factory filters and the Talcott study around 18 percent.
Q. Are each of these studies discussed in greater detail in your report, sir?
A. They are.
Q. I'd like to return to Quebec. By 1997 was there an

important update on the Quebec cohort?
A. There was.
Q. And what did it show?
A. So the researchers noticed that there was a difference between the number of mesothelioma cases in the town of Asbestos which was the largest mining community in Canada and the town of Thetford which is a smaller mining community.
Q. Okay. And what did they find was the usual explanation --
A. So --
Q. -- in asbestos?
A. -- not only was asbestos mined in Asbestos, but there were also -- there was also a crocidolite manufacturing factory in the town of Asbestos and there was a lot of work back and forth between the factory and the mine.
Q. All right. And so they had some excess cases in the town of Asbestos, but those were attributed to what, sir?
A. To the crocidolite factory.
Q. Okay. And now, the other town was the town of Thetford Mines.
A. Right.
Q. It was a different mining region in Quebec, right?
A. That's right.
Q. And were there excess cases of mesothelioma there too?
A. There was. And what was important, I think, about that

finding is that there was a difference even within Thetford between the centrally located mines and the more peripheral mines.

Q. And is this an historic diagram that shows the difference between where the central mines and the peripheral mines were?

A. It is. It shows the physical relationship of the mines.

Q. And what was the conclusion of the McDonald group with regard to that difference?

A. So the group led by Dr. McDonald found that there was really little or no evidence of increased risk for mesothelioma in the peripheral mines. And their hypothesis at the time is that the central mines, because they had a higher relative content of tremolite, that it was in fact the tremolite that was causing the excess number of cases that they were seeing in relationship to the central mine.

Q. Okay. Now, I think there's reference in the briefing to the articles by Dr. Lemen and his co-author Stayner when in the late 1990s they surveyed the world literature to try to find every chrysotile-related case they could and what, four-fifths of them or something were all from this one cohort?

A. That's right.

Q. So mesothelioma in chrysotile was rare. The place it occurred the most was in Quebec. And once the final studies were done, it was clear that in some mines, what was the real

cause?
A. Tremolite, an amphibole type fiber.
Q. I'd like to return to South Africa. That's where our story of the history began. Has there been significant research done in South Africa on the issue of the difference between the fiber types in causing mesothelioma?
A. There has. And I pulled out two studies by Dr. Rees and Dr. White in 2001 and 2008 to demonstrate my point.
Q. Okay. And what do those studies demonstrate?
A. So South Africa not only had crocidolite mining done, some amosite mining done, it also had quite a bit of chrysotile mining done. In fact, as the paper indicated, there was large work force and production of a hundred thousand tons per year of chrysotile, yet they saw no mesothelioma cases detected in these South African chrysotile workers.
Q. Okay. And that heavy chrysotile mining has been going on since the decade of what, the 1920s?
A. I think it was in the '20s, yeah.
Q. Okay. Plenty of latency for mesothelioma to show up?
A. Yes.
Q. And they weren't getting it by the miners in the same country that mined amosite and crocidolite where there is a high rate.
A. That's right.

Q. All right. Was there also a series of studies that -- back up in Canada different than the McDonald cohort that looked at women who happened to live near the two mines we're talking about, the mine in Asbestos and the mine in Thetford Mines, right?
A. Yes.
Q. Tell us about this.
A. This focused on the asbestos in Thetford Mines again looked at non-occupational type of exposures. And what they found is that, again, there was a difference between the two mines. So in Asbestos, the town of Asbestos, there were no cases found. Yet in the Thetford mine there were ten cases found and all were associated with proximity to the central mine not to the more peripheral mine.
Q. Just so that we understand what the proximity meant, these were people that lived right near the mines, correct?
A. Yeah. When I say "proximity," I mean proximity. They were, in fact, living right next to the mine.
Q. All right. And so people living right next to the mine in Asbestos, the largest chrysotile mine in Canada, those women, they didn't have any excess cases?
A. No excess cases.
Q. But in Thetford Mines, the other city, that's where it was, right?
A. That's right.

Q. And that corresponded with what the other cohort had found, the McDonald cohort?
A. That's right, the occupational cohort and the non-occupational cohort matched up well.
Q. Well, what does that tell us about the chrysotile that's imported from Canada? Does that mean all chrysotile is or isn't contaminated --
A. No.
Q. -- at the high levels that can cause this problem?
A. No, I don't think you can make that kind of blanket statement. I think it's clear that some chrysotile has some tremolite contamination at the mining source and some doesn't.
Q. All right. We're in 2013. We've made it that far in the history of our lives. Can you tell us -- can you fill out your chart.
A. So the chart has then been filled out by studies that have occurred in the last decade. There's been a maturation of the more historical cohorts and the mesothelioma deaths listed here, but there's also been additional information that's come from other studies as well.
Q. All right. Now, some of these studies I wanted to focus on a little in particular.
You've included on your list the Loomis study in 2009.
A. Yes.
Q. The Loomis study.

A. Yes.
Q. And that's been a study we've talked about a little in this case. It involved how many plants?
A. Four all together.
Q. And one -- and in the study itself it mentioned that one of those plants used amphiboles. That was plant 3, right?
A. Correct.
Q. And then there was a comment in that study about plant four, the Marshville plant, that said what?
A. That there was essentially no amphibole exposure in that plant.
Q. Okay. And have you looked at the documents from that?
A. Yes.
Q. And what's the real story there?
A. I think it's reasonable to assume there was amphibole exposure in that plant.
Q. Okay. What about the other two plants? Was there even a suggestion that the other two plants that for years used only chrysotile here in North Carolina, was there any mesothelioma in either of those two other plants that even could possibly be attributable?
A. No. Plant 1 and 2 did not have any mesothelioma deaths.
Q. Okay. So on this chart, when you mention zero rate, it's uncontroverted that for at least two plants, there's zero mesothelioma in them, right?

A. That's right.
Q. Now, if we have just one study where mesothelioma isn't shown in a population, does that necessarily mean that the agent we're investigating can't cause mesothelioma?
A. No. I think when you're making these sorts of causal determinations, it's important to consider the whole range of medical literature that's available to us. And now we have the benefit of having 50-plus years of studies in this area.
Q. All right. And based upon the currently existing medical literature. Based on knowledge in 2013, not 1972, is it reasonable scientifically to say that the epidemiology demonstrates that chrysotile fibers cause mesothelioma?
A. I don't think that's reasonable.
Q. Even including tremolite, if you want to include tremolite contaminated or other contaminants, where are the only populations where an excess rate of mesothelioma is being demonstrated?
A. In the mining community. So at the source of the chrysotile itself.
Q. Okay.
A. And which should always be distinct, and we should always make sure we draw that distinction between that and the end user?
Q. So in a mining community, these people -- we've heard about fibers per CC years, and Mr. Henshaw's chart gives us

fibers per CC years numbers.

What kinds of cumulative lifetime exposures are miners exposed to in mining any of these asbestos minerals?

A. So in the Quebec mining community, for instance, we're talking about several hundred fiber years of exposures. And in the Italian chrysotile mining cohort, we're also talking on the order of hundreds of fiber years. So quite a bit of dose.

Q. All right. Anything like that dose implicated even arguably for gaskets and packing?

A. No, one can't argue that.

Q. And now, you mentioned that mining populations have a high exposure. Are there some populations where it has been argued that in manufacturing where people daily are working with the raw materials to make the products, that there may be some cases that could be attributable to the chrysotile?

A. There's been that argument.

Q. Does that exposure equal in quantity the nature of the exposure someone would have as an end user, episodically changing gaskets or doing something like that?

A. No.

Q. Sir, the next topic I'd like to turn to is something that Mr. Henshaw was asked about. The words were -- when it was shown in the regulatory literature, he said yes, that's based on a linear model, no threshold model. Can you tell us what that really is.

A. Sure, and I'm going to use the assistance of a slide.

So the first concept that has to be understood, and this is very standard dose-response curves.

So the basic idea is is that the number of cases of any disease goes up as the exposure amount increases. So whether or not we're talking about aspirin, alcohol. I know there are only nondrinkers in here; but if we talk about alcohol in that way, if we talk about asbestos, silica exposure, anything can be very safe at a low dose but kill you at a high dose. Even aspirin, even coffee, of course, even alcohol.

Q. Okay. And so you've shown us here a sort of standard dose response curve. Why does it have this S shape?

A. The S shape comes in because the dose can increase to the right here on the horizontal axis, but that doesn't necessarily mean that the number of diseases increases or the number of cases increase. And that introduces the concept of a threshold.

Q. Okay.

A. So a threshold is simply an amount of exposure that above which there is no elevation in disease risk -- I'm sorry, below which there is no elevation of disease risk and above which there is.

Q. Okay. And just to be clear, when we say there is no known safe level, do we yet know exactly where the thresholds are for asbestos?

A. Well, there's always a lot of conversation about the words "no known safe level."
Q. Yeah.
A. That's -- the known part of that is a very important word because there's a difference between being able to comment that there is a threshold versus knowing exactly where it exists. So the fact that there is no known threshold certainly shouldn't be interpreted that one doesn't exist.
Q. All right. And in fact, for almost every substance that's ever been investigated enough, sooner or later if you do enough enough work, you'll find those thresholds right?
A. That's right.
Q. Now, we've heard about public health agencies or regulatory agencies like OSHA --
A. Yes.
Q. -- like EPA, and that they have done risk assessments. And can you explain how the assumptions are made scientifically in areas where we don't have data.
A. So this is again a dose response curve or a dose response chart. So the dose on the bottom, moving to the right increasing; the number of cases on the vertical axis increasing.
And so what you've got is two very distinct zones. One is called the zone of observation. So the zone of observation becomes the zone of observation because you have actual data

to put into that chart.
Q. And where is the data represented in this hypothetical, very hypothetical chart?
A. So these are hypothetical data points where you have essentially got high exposure and you've got a high incidence of disease.
Q. Okay.
A. And if you relate this to the asbestos story, that's where the medical literature informs us. These historical cohorts that had high exposure, high rates of disease.
Q. I see. Well, could you tell the court how public health agencies are instructed to and how they do go into the areas where they don't have an observation data.
A. So in not being critical of what they're doing, the regulatory agencies, they're doing something else. They by necessity have to deal with a zone of inference. And the zone of inference simply means they don't have data points to fill in down here at very low doses. They simply don't have the data to make a firm conclusion about, so they have to infer that conclusion and that's why it's called a zone of inference.
Q. And how do they do that?
A. Well, what's been decided from a regulatory standpoint is to simply draw a straight line back from the zone of observation where the actual data exists all the way down to

zero. And I think you heard Mr. Henshaw talking about the linear no threshold model.
Q. Right.
A. That is the linear no threshold model. You essentially go from what's been observed at high doses and draw a straight line all the way back down to zero.
Q. And does that mean that it is scientifically established that cases will occur on that straight linear model line?
A. No, not at all.
Q. And the true dose response curve may look like that?
A. Sure.
Q. Or that or anything?
A. It could look -- it could look like either one of those depending on the substance that we're talking about where, in fact, at low doses of exposure, there is no excess risk of disease.
Q. And indeed, the EPA, for example, did a risk assessment in the '80s, I think there's a lot of talk about. When that risk assessment has been compared to actual study populations 10 and 20 years later, has it been found to accurately increase, predict the number of cases or not?
A. No, I think it's safe to say that that model overestimated the risk of disease.
Q. All right. But from a public health standpoint, is that bad?

A. No, not necessarily. I think they're charged with being conservative in the interest of protecting the public health. And their mission is more of hazard identification rather than establishing the kind of causal relationships that I think we're interested in here.

Q. All right. Sir, so the regulatory model is a straight line model.

In dealing with the OSHA model, what studies were used -- you said that the data used to make this -- these models back in the '80s was older data. What studies were used?

A. So I've listed the six studies that were actually used to be put into the OSHA model. And I'd also draw your attention to the cumulative exposures that were within those cohorts. And you can see that even at a minimum, the studies used contained 55 years of exposure all the way to a maximum of 600 years of exposure.

Q. And we can see the kinds of exposures that occurred in these. Like Selikoff's study, what's the cohort size? 17,800. Is that the -- what is that, the insulators?

A. Those are the insulator cohorts. Those are the number of people he studied in his cohort.

Q. And would it be fair to say 25 to 75 percent of that was amosite?

A. Yes.

Q. Your name is on here. In 1979 were you doing these

studies?
A. I was actually a tenth grader, I think, at that time. So that was not me; that was my father.
Q. All right. And in that study, tell us a little about it because we don't have the fiber type there.
A. So the -- my father and his group actually studied two different cement factories outside of New Orleans. Both involved chrysotile exposure, but one had a much heavier exposure to crocidolite as well.
Q. So that was a plant that also used crocidolite?
A. Yes.
Q. Okay. Were the cases there attributable to the chrysotile or the crocidolite?
A. The cases occurred in the crocidolite-exposed people but not in the chrysotile-exposed people.
Q. All right. So those were the studies basically that were actually put into the regulatory model, right?
A. That's right.
Q. They don't tell us -- we didn't have data. The zone of inference here would be below 55 CC cumulative exposure fiber years.
A. That's right.
Q. Sir, we've heard something about potency. What do we mean when we're talking about fiber potency?
A. So fiber potency is the notion that I've talked about

where there is a difference in the propensity of certain fiber types that cause disease. And that's what the evolution of this medical literature has provided for us: More information about the different fiber type potency.

Q. All right. And can you, addressing the court, tell us what the modern quantitive literature has told us about fiber types, sir.

A. So there's been two large studies that have looked at the quantitive relationship between amphibole exposure like crocidolite and amosite to chrysotile exposure. And the most probably widely quoted one is the one in 2000 by Hodgson and Darnton that looked at a number of cohorts with quantitative exposure information and the incidence of disease and found that the mathematical relationship between crocidolite, amosite, and chrysotile was 500 to 100 to 1.

And it's important, I think, to point out that when the authors discussed chrysotile exposure, in this setting they were talking about not the end user but chrysotile miners, assuming that there was some tremolite contamination in that group.

Q. So even assuming that you include the tremolite -- the cases that were tremolite related, the fiber potency that Hodgson and Darnton came up with was 1 for chrysotile, 100 for amosite -- and we've got it in brown. Is it called brown asbestos sometimes?

A. Yes.
Q. And then crocidolite sometimes called blue asbestos?
A. Right.
Q. And that's 500. That's the most potent.
Is there another peer reviewed published quantitative assessment of the relative potency of the fiber type?
A. There was also a quantitative analysis done by Berman and Crump published in 2008 that looked at a variety of metrics that could either mathematically be rejected or accepted. And one difference with Hodgson and Darnton is Berman and Crump also used fiber size in their models as well and found that either the notion that chrysotile elevated the risk of mesothelioma which was rejected entirely by some of the metric studies or it was exceedingly uncommon. And the relationship then drawn between amphibole exposures was in the neighborhood of 900 to 2000 times more likely for amphiboles to cause mesothelioma as opposed to chrysotile which was either zero or one depending on the metric studied.
Q. Now, there's been -- as we sit here today in 2013, is there anyone that in the peer reviewed literature really denies that there's a substantial potency difference between the amphiboles and the chrysotile?
A. No.
Q. Now, there's some old literature that expressed opinions about that, right?

A. Yes.
Q. All right. And we'll get to that in a minute, but if you factor in the potency difference into the OSHA studies, does that help us get a better understanding of what the true dose response curve is?
A. Yeah. If you plot the OSHA studies and include ambient air, for instance, or the Canadian women's study or the Canadian men's study that I talked about earlier, you've got this sort of dose relationship. And what this did -- what this slide depicts is the notion that there was a certain chrysotile content within those OSHA or EPA studies. They were mixed studies. They were neither pure amphibole or pure chrysotile.
And so if you plug those potency factors in to the total cumulative dose that was established in those studies, you've got a dose response curve to chrysotile that looks something like this.
Q. And that resembles the S curve that is the normal dose response curve.
A. Very much so.
Q. All right. Sir, on the other fiber studies, we heard about a publication in a Japanese journal in 2001 by Dr. Nicholson. Are you familiar with that paper?
A. I am.
Q. And have you commented on that paper in your report in

detail?
A. I have. In my rebuttal report I go into some detail about it.
Q. And that was because it was cited by the committee experts?
A. That's right.
Q. And Dr. Nicholson in 2001 talked about -- he agreed that crocidolite was much more potent, but talked about similar potencies for amosite and chrysotile?
A. For amosite and chrysotile, yes.
Q. And he had been the person who had done the risk assessment back in the 1980s --
A. Right.
Q. -- right?
And when he did that risk assessment way back in the 1980s, what did he assume about potency?
A. He assumed at that time that the potencies were equal between all chrysotiles.
Q. Okay. And 2001 -- actually, the paper was submitted in 2000. Was there -- had he -- well, I'm sorry. You've looked at the paper. Did he cite new data? Did he do a new quantitive risk assessment? Did he go through all that?
A. No. It was mostly the 1980s data once again represented and very much using the regulatory model assumptions that we talked about earlier.

Q. I see. And he used three different approaches. Briefly can you articulate what his approaches are and why --
A. Yeah.
Q. -- they don't hold up based on the modern data.
A. The basic approach was is that all the asbestos type fibers were equally potent in causing lung cancer, not mesothelioma but lung cancer. And he used then lung cancer as a surrogate measure of cumulative asbestos exposure. And he thought by doing so, you could look at the lung cancer/mesothelioma ratio and unmask the fiber differences using that strategy.
Q. Does that work?
A. Not really. Lung cancer is multi-factorial. It's not purely related to dose, although dose is certainly an important parameter.
Q. Yes, sir.
A. It's not entirely related to that. It would be related to the development of fibrosis in an individual. It would be related to the length of follow up, whether they smoked or didn't smoke.

So in my view you can't just take lung cancer incidence in an asbestos-exposed cohort and use that as a surrogate for asbestos exposure.

Q. And then in that method, he used eight studies, but as your report, I think, explains, the eight studies and the data

from those eight studies were the same data he was using in the '80s for his risk assessment, right?
A. Very much so. And I think the important fact is those studies were confounded as well.
Q. What do you mean by confounded, sir?
A. That there was an amphibole component to those exposures. So although I think there was an attempt to classify them as pure chrysotile exposures, they really weren't. They really were confounded with an amphibole type exposure which we've talked about is a very important confounder.
Q. Okay. The second method he used was something called a time course and what was wrong with -- or what has come out in the literature about the assumptions that Dr. Nicholson was making?
A. Dr. Nicholson assumed that there was no amphiboles used before 1937.
Q. In the United States?
A. In the United States. And by doing so, he was essentially starting a latency clock in 1937 in order to do his modeling of disease incidence.
Q. Okay.
A. Now, there is not a lot of good evidence that that's the case. In other words, there was amphiboles used before 1937.
Q. Uh-huh.
A. And that was documented in the Langer publication in 1998

as well as a discussion in the Hodgson and Darnton paper in 2000.

Q. And since the 2001 paper, have there been peer reviewed published documents that have further shed light on this issue?

A. I think so, because I think that further publications by Price and Ware in 2009 and by my father in 2004 show that there is a very close relationship between the amphibole consumption peak in this country and the number of mesothelioma cases showing up after an appropriate latency period.

Q. Okay. And so if, let's say, the asbestos control procedures that went into effect in the '70s were ultimately effective, is there any reason to believe that there are going to be mesothelioma cases attributable to asbestos exposure that's going to happen after that?

A. No. I mean, in my view and the view of some of the modeling that's been done in these publications, there's going to be a diminishing number of mesothelioma cases for a period of time that is going to correspond well with the peak in amphibole exposure that we've seen -- or amphibole consumption that we've seen.

Q. Thank you. Last topic is your view on gaskets. Are there epidemiological studies on a population that is exposed to higher levels of asbestos, chrysotile asbestos than gasket

workers that can enlighten our view on whether gaskets are a cause?
A. Yes.
Q. What population is that?
A. This is in the auto mechanic population. The auto mechanic is another low dose asbestos kind of exposure where there's been a number of case control studies, some of which I have put on this slide that show no elevation in risk in this occupational cohort.
Q. All right. And that's -- Mr. Henshaw explained to us that the level is somewhat higher from brake mechanics than gaskets; is that correct?
A. That's correct.
Q. And yet still, there's no exposure -- no increased risk.
A. That's right.
Q. Dr. Selikoff wrote that there is no health hazard in forms used in shipyard applications for gaskets and packing, compressed asbestos sheet packing.
Do you agree or do you disagree in a more general sense with the use of gaskets?
A. Yeah, I think that that's what was known in the late 1970s when Dr. Selikoff wrote that.
Q. So in terms of just whether chrysotile-containing compressed asbestos sheet gaskets can produce enough exposure to cause mesothelioma, do you have an opinion based on the

peer reviewed published literature informed by case control studies? Do you have an opinion on that?
A. I do.
Q. What's the opinion?
A. I don't think that these sorts of exposures to low dose chrysotile elevate the mesothelioma risk.
Q. And in addition, Garlock made packing.
A. Yes.
Q. And the information we've seen on packing has -- what's your understanding of the level of exposure from packing when it compares to gaskets?
A. Very low. Very low.
Q. It's even lower than gaskets?
A. That's right.
Q. Does packing -- exposure to packing contribute to cause mesothelioma in anybody?
A. No.
Q. But there's a broader issue that the law defines for us and that is this issue of specific causation by comparative dose. And you have reviewed the material provided by Dr. Henshaw -- Mr. Henshaw; is that correct?
A. Yes.
Q. And he has examined many sources of exposure that is likely for claimants that will exist now or that may exist in the future; is that correct?

A. Yes.
Q. And based on your knowledge of occupational -- of these occupations of the people you've seen with mesothelioma, the work you've done, the literature, has he looked at the right kinds of exposures?
A. I think so, yes. I think he's been comprehensive in that way.
Q. And you mentioned in your report that he's made conservative assumptions. Why do you say it's conservative or what's one implication of that?
A. Well, I think that the main reason is that he also took out letter F here. He took out the exposures that would be directly handling insulation during tasks that weren't related to work involving gaskets and packing.
Q. And what do you mean by -- what would that be?
A. That could be just removing insulation that's not related to actual work with gasket and packing or doing some other kind of work that isn't directly gasket and packing work but is resulting in an amphibole type exposure.
Q. Okay. So here were his conclusions by four example occupations within four groups.
For group 1, is the gasket exposure when compared with the total lifetime exposure likely for people in that group, is that a substantial cause of mesothelioma?
A. No. And this slide looks at the total asbestos exposure

without regard to fiber type and looks at the comparison between the amount of exposure one gets with an insulation type product versus the amount of exposure one gets from a gasket and packing.

Q. So this -- your opinion on this, is it just in raw terms? Doesn't matter about fiber type?

A. No, I didn't put fiber type into this slide.

Q. Okay. And Mr. Henshaw didn't put fiber type into the slide.

So in your view, is the exposure in group 1 occupations -- in any of the groups, a significant contributing cause or substantial cause of mesothelioma when considering the total likely lifetime exposure of people that will be in these groups?

A. No.

Q. Now, you've made the point that you've excluded the fiber type issue. Scientifically should we be excluding the fiber type issue?

A. No, not at all. As I tried to point out over the last hour, the fiber type is a very important issue.

Q. Well, if we do what science would require, which is to look at the difference in fiber type, fiber potency difference of 500, 100, and maybe even 2000, if you take that and put it -- let's say even with the highest group, this group 1 on the pipefitters, how graphically could we represent taking

into account the potency difference?
A. So if you assume that the insulation that we're talking about contained about 50 percent chrysotile and about 50 percent amosite, and you assume that amosite was about a hundred times more potent than chrysotile, we can demonstrate that by fiber type there's quite a big difference between these kinds of exposures.
Q. So how would we have to alter this diagram? Have you animated this diagram to show us --
A. Yes, I've animated it.
Q. Well, you've animated it, but it hasn't animated. The best laid plans.
Here you go.
A. So if you take into account not just the total amount of asbestos exposure but what kind of fiber they were exposed to, the differences between the exposure quantity from the insulation products is going to be very much different from the gasket and packing products.
Q. So the gasket and packing products remains the same, but you couldn't show it on the previous scale, right?
A. That's right.
Q. So you'd have to multiply the insulation exposure by some factor to take into account potency to be scientifically reliable.
A. That's right.

MR. SCHACHTER: Thank you, Dr. Weill.

MR. GEORGE: Your Honor, if you don't mind, can we take our afternoon break just a little early so I can get ready?

THE COURT: Sure. Let's take a break until ten minutes after 3:00.

MR. GEORGE: Thank you.

(Brief recess at 3:00 p.m.)

THE COURT: All right. Mr. George.

CROSS EXAMINATION

BY MR. GEORGE:

Q. Good afternoon, Dr. Weill.

A. Good afternoon.

Q. This is not our first encounter, correct?

A. No. I'd rather call it a meeting than an encounter.

Q. We had a meeting --

A. Yes.

Q. -- in October in Los Angeles in a stucco case, correct?

A. Yes.

Q. So you know there's another side to the story than what you just presented, correct?

A. I do.

Q. Okay. You first began testifying in lawsuits in 2002, correct?

A. Are you talking about asbestos lawsuits?

Q. Any lawsuits.
A. It was actually a bit before that. I testified on behalf of plaintiffs in the fen-phen litigation.
Q. You've given about 150 depositions.
A. That's about right.
Q. You testified six times in asbestos cases in trial and you testified twice in two different bankruptcies, correct?
A. A bit more now in the asbestoses cases. I think it's 10 or 12.
Q. My updated list only had six, but it's now 10 or 12?
A. Yeah, I think so.
Q. And you were just deposed in an asbestos case about a week ago, correct?
A. That's probably about right.
Q. For a case in Kentucky?
A. Yes.
Q. You've never testified in trial or in a deposition on behalf of an individual claiming injury from exposure to asbestos, correct?
A. That's correct.
Q. Let's talk about your compensation very quickly. You charge $500 to review records and/or to write a report, correct?
A. Yes.
Q. And then $600 to come to court to testify about what

you've written, right?
A. That's correct.
Q. All the fees go to you. They don't go to Stanford.
A. That's correct.
Q. And you don't sit here as a representative of Stanford. None of the opinions you offer today are offered on behalf of the university, correct?
A. No, I don't.
Q. Over the years from 2002 to the present, projecting through 2013 you've made over $4.5 million as a litigation expert, correct?
A. I think that's a fair estimate.
Q. And these are numbers -- beginning about 2005 you started getting into about the 200 to 250 thousand dollars. Did that for about four years. And then it's pretty much gone up every year since. It was 400 in 2009, 600 in 2010, 800 in 2011, 850 in 2012, and you think you're going to do even better than that this year, correct?
A. I think that's probably right.
Q. Now, Garlock's paid you $227,650 for the effort that you put forth to offer your opinions in this case, correct?
A. I've never totaled it up, but I did give you guys the invoices for the depositions. I'll accept that.
Q. Provided my math is correct, this is a good number.
A. Provided that is correct.

Q. Now of that time, 30 hours of it was spent talking to the lawyers for the debtors, Mr. Schachter and Mr. Harris.
A. That's right.
Q. And that doesn't include the fact that at least on four occasions in July, August, September, and December you had meetings, but you didn't break out how much time those meetings took, correct?
A. That's correct.
Q. So that's about $14,000 of the money you earned was just talking to the lawyers about your opinions.
A. I'll accept that.
Q. And these are opinions that you've been offering for the last decade, correct?
A. I think some of them, but not all of them.
Q. Your qualifications, you're not an epidemiologist.
A. Correct.
Q. We heard from an epidemiologist so I'm not going to talk to you about the epidemiology of brakes because we had that discussion with Dr. Garabrant. And you wouldn't have anything to offer this court any more than Dr. Garabrant did.
A. I don't think so. I think he's quite qualified to give those opinions.
Q. You're not an industrial hygienist. We've already had three of them so I'm not going to ask you too many industrial hygiene questions.

You're not an expert in occupational medicine, and there is a field where you can be certified to be an expert in occupational medicine, correct?
A. That's correct.
Q. You can be board certified as an occupational medicine specialist and that's not what you chose to do, right?
A. That's correct.
Q. You've never written an article on mesothelioma.
A. No.
Q. You've had one letter to the editor that deals with non-malignant asbestos disease.
A. Partly. And partly with mesothelioma as well.
Q. Your two book chapters dealt with the diagnostic and clinical features of asbestos disease but not the causation of meso, correct?
A. I don't know how deeply I got into causation in those chapters. I haven't read them in some time and I'm not sure if I touched on that or not. But I would accept they're primarily associated with the clinical aspect of the disease.
Q. You certainly didn't go into any depth about your views on whether chrysotile can or cannot cause mesothelioma, correct?
A. I did not.
Q. And then you have one published article on non-malignant asbestos disease that can or may be caused by exposure to

vermiculite, correct?
A. That's correct.
Q. Neither of the senate testimonies that you've given address the issue of whether asbestos causes mesothelioma, correct?
A. I think the second senate testimony did touch on that.
Q. I deposed you -- actually, I took your trial deposition and I think I asked you the very same question.
You certainly didn't tell them -- offer the opinions that you offered to the extent you've offered them today.
A. No, I didn't.
Q. You've never consulted with any government agency where you stated your opinion that only extremely heavy exposures to chrysotile could cause mesothelioma, correct?
A. Correct.
Q. And you've done no original research on your own on mesothelioma.
A. That's correct.
Q. Now, you would agree that exposure to chrysotile can cause pleural plaques.
A. I think in the proper exposure setting, yes.
Q. And pleural plaques are a benign scarring in the lining of the lung.
A. That's correct.
Q. Same place where mesothelioma develops.

A. That's correct.
Q. So in order to cause pleural plaques, the chrysotile fibers have to get through all those defense mechanisms that you talked about, then get translocated into the lymphatic system and get to the area of the pleura where they then reside long enough to start the process of scarring, which is a pleural plaque.
A. Well, my opinion, though, regarding chrysotile asbestos, and you're using that term fairly generically, but when I say that it can cause pleural plaques, I'm really talking about the amphibole component of that and that's why I wanted to be sure -- it would have to be an exposure setting that I think would elevate the risk for plaque development.
Q. But you would agree that exposure -- do you agree that exposure to chrysotile fibers without any contamination can cause asbestosis?
A. I don't think it can. So in settings where we're only talking about chrysotile itself, I don't think that it's been demonstrated that there is an excess risk of asbestosis just because of the lung biologic reasons I outlined earlier.
Q. So if I understand your opinions, you don't believe that exposure to chrysotile, the fiber itself, without any trace contaminants or co-mineralization of an amphibole can cause either pleural plaques, asbestosis, lung cancer or mesothelioma, correct?

A. Not the pure fiber itself, no.
Q. So you think the pure fiber which made, what, 95 percent of the asbestos that was used in the United States --
A. Yes.
Q. -- is totally innocuous?
A. I think so.
Q. Unless you're an asbestos miner.
A. I think if you're an asbestos miner with very high doses and there's known tremolite contamination, so those two instances have to be present.
Q. So you believe that chrysotile if it has tremolite in it can cause lung cancer but it's really the tremolite doing it and not the chrysotile, correct?
A. Yeah, and that's why I think there has to be a lot of attention paid to when one just talks about chrysotile asbestos, really what exactly are you talking about? Are you talking about chrysotile that is known to be contaminated with tremolite? Are you talking about chrysotile at the source in the mining communities? Are you talking about chrysotile at the end product?
Q. Would you agree with me that there are very few, if any, cohorts of workers who have been exposed to pure chrysotile?
A. I don't think there are many.
Q. Okay. And that's -- that includes all end product users, correct?

A. Yes.
Q. Even end product users that are in environments where they're using chrysotile products, the likelihood is that in making that product, there is some trace tremolitic contamination.
A. That's the likelihood, yes.
Q. Okay. So it really doesn't matter, then, to you whether it's a pure chrysotile exposure or not because it just doesn't exist is in the real world.
A. Well, I wouldn't say that. I think if you're asking me as a physician whether these kind of exposures can be considered pure chrysotile exposures, from a technical industrial hygiene setting perspective, the answer is no.
But if you're talking about such minute amounts of amphibole contamination, because impurities happen in all substances, from a physician's standpoint, it doesn't make any difference because those kind of exposures don't elevate the risk of disease.
Q. But in your opinion that's true for every time you're confronted with a chrysotile-exposed individual. You've never testified in a joint compound case that despite the fact that the person could have done the job for 30 years with a product that contains up to 7 percent chrysotile asbestos, that that exposure ever was a contributing cause to their mesothelioma, correct?

A. I don't think it does.
Q. So basically, the only time that you think that chrysotile asbestos can cause mesothelioma is if you were a Canadian miner.
A. A Canadian miner. And I think the risk has been elevated as well in the Italian mining cohorts.
Q. We'll talk about that in a little bit.
Now, these organizations disagree with that conclusion, correct? All of these organizations, the Canadian Medical Association, the American Public Health Association, the American Cancer Society, the World Health Organization, all of them stand for the proposition that any asbestos fiber type, contaminated or not, can cause mesothelioma, correct?
A. I think that the associations say a lot of different things. They certainly recognize that there's fiber type potency differences. And if those statements are that all fiber types can cause mesothelioma, I would then look to the next sentence to see if the cohorts they're talking about are the same cohorts that I'm talking about.
Q. But --
A. And that's why I think it's difficult to take statements such as these and make a blanket application to all kinds of chrysotile exposure.
Q. Except that you don't know of any statements from any organizations where they've agreed with you and said the only

exposure to asbestos that doesn't cause any disease is pure chrysotile?

A. No, I'm not aware that that statement has been made.

Q. And you're aware that these organizations, the Canadian Society for Epidemiology and Biostatistics, the International Epidemiologic Association, the American College of Epidemiology, and the National Academy of Sciences, they all make the same type of statements that every type of asbestos --

MR. SCHACHTER: Your Honor, may we approach the bench, please? Well, I guess we don't have to approach the bench, we're not in front of a jury.

He just violated an order, being clever like they try to do. We had -- we tried to do discovery into this joint policy statement by the Society of Epidemiologists written by a non-epidemiologist and the court said you don't get to do that discovery, but they don't get to mention it. And because these agencies have signed on to that, they're now putting that up before the court.

These are just not -- you know, he's violating the court order. If we had done the discovery to show who actually wrote those things and how they were solicited, we would have shown the nature --

THE COURT: All right. We'll sustain the objection.

MR. GEORGE: Your Honor, none of this -- just to

defend myself. None -- this is from the deposition that he just took two weeks -- a week ago. None of that has to do with the Societies of Epidemiology. He was asked about specific organizations and what their position was. Says nothing about Societies of Epidemiology. Went through all of the various organizations and that's what his response was. I wasn't referring to any policy statement.

MR. SCHACHTER: This is indirect. That's where they have signed on to that policy statement and of course he acknowledged that.

THE COURT: Let's go on to something else.

BY MR. GEORGE:

Q. There are other organizations like the National Toxicology Program, the United States Public Health Service, and the World Trade Organization that all disagree with your constricted view of chrysotile as a cause of mesothelioma, correct?

A. I don't know what each one of these says specifically about it. All these documents say a lot of different things and so I'm not really sure exactly what you're referring to.

Q. But you did just testify last week that those organizations all agreed that all asbestos fiber types, including chrysotile can cause mesothelioma.

A. In parts of their document they say that and in parts of their document they say other types of things. So I don't

want to endorse everything they said in the document.
Q. Well, the World Trade Organization when there was a dispute, when Canada was attempting to sell chrysotile to France and France didn't want it, the panel, after noting that the carcinogenicity of chrysotile asbestos fibers has been acknowledged by international bodies and confirmed by experts, the panel consulted ruled that it has sufficient evidence that there is in fact a serious carcinogenic risk associated with the inhalation of chrysotile fibers. In fact, the scientific evidence of record for this finding of carcinogenicity of chrysotile asbestos fibers is so clear and voluminous and is confirmed a number of times by a variety of international organizations so as to be practically overwhelming.
That's what that panel found, right?
A. That's what it says.
Q. You agree 95 percent -- now, what that means is when we are in a shipyard situation like what's up here, you would agree with me on those pipes, what we have is chrysotile asbestos cloth that's wrapped around the insulation, correct?
A. Yes.
Q. Sealed with chrysotile asbestos cement, correct?
A. Well, I think now we're getting into generalizations about what was present in these kinds of facilities. I agree when Mr. Henshaw testified this morning that it's very difficult to make generalizations about all the kind of

exposures that are out there. And so I would be reluctant to do so.

Q. But since 95 percent of what was used in the United States was chrysotile, that would mean 95 percent of the asbestos that's in that engine room is chrysotile, correct?

A. I wouldn't jump there because just to say that there's 95 percent chrysotile asbestos as a total consumption in the U.S., it doesn't say what's in a particular exposure setting or not.

Q. Let's talk about animal studies because that's something that Dr. Garabrant would not -- didn't have the expertise.

You agree that it's been demonstrated that pure chrysotile, I mean, uncontaminated chrysotile can cause mesothelioma in rats.

A. I'm not aware of any study that looked at pure chrysotile and found mesothelioma. I'm aware of some that don't, for instance, by Ilgren and Muhle and colleagues.

I would also, and didn't have time in my direct to talk about the fact that in rats, you can essentially, if given enough dose cause tumors of a variety of sort from a variety of different exposures, including to glass or to wool or to cellulose.

So the animal studies can be used to make certain mechanistic conclusions, but I definitely wouldn't make causal associations based on animal studies alone.

Q. You would agree that a good scientist will evaluate the totality of the scientific evidence before making a decision about causation.
A. Yes.
Q. That includes animal studies, in vitro studies, epidemiologic studies, case reports, and any other industrial hygiene studies about dose, all of those are important pieces to the puzzle of does something cause something, correct?
A. Well, there's a couple things in what you said. One is that that process of evaluating the totality of multi-disciplinary approach is a risk assessment approach.
I think what your question implied is that animal studies are part of the causal association determination, and they're not. That's where epidemiology comes in.
Q. So you think that the scientists that do this type of work are wasting their time?
A. No, not at all. Not at all. As a matter of fact, I think there are important mechanistic findings from animal studies that have helped us in a variety of ways in medicine. So I don't think it's a waste of time at all.
Q. When these scientists who do these animal experiments experiment with the substance, they typically use what is known as the UICC samples, correct?
A. Yes.
Q. And you're aware that Dr. Dodson and Dr. Frank took the

UICC sample A for chrysotile from Canada and they looked at twenty some thousand consecutive fibers and found no evidence of tremolite. They published that study.

A. I think UICC-B is from Canada, if I'm not mistaken.

Q. Well, we'll see what the article says.

A. Yeah, I know which article you're talking to. I haven't reviewed it in some time.

Q. But you will agree that the use of chrysotile in animal inhalation experiments with rats have caused mesothelioma.

A. Yes. I mean, if you look at the animal studies that I outlined in my report, the propensity of chrysotile to cause tumors in rats is under a couple different circumstances. One, that you have to give the rats a very high dose and you have to give it for a long time.

I think the reason I included the animal studies in my report is to show that it's much easier to make mesothelioma in a rat giving them amphibole than it is chrysotile. And I don't think that's a debatable point.

Q. Well, let's look at the studies. First of all, you're familiar with the IARC, the International Agency Research on Cancers, 2012 publication on asbestos, correct?

A. Yes.

Q. And in that publication, they went through all the rat studies and other animal studies and said, bronchial carcinomas and pleural mesotheliomas have been observed in

rats after exposure to chrysotile, crocidolite, amosite, anthophyllite, and tremolite fibers, correct?
A. Yes. And in that bland statement, as I mentioned, I couldn't disagree with that.
Q. Now, this is 1974, J.C. Wagner, the guy who in 1964 -- or 1960 had found the case of mesothelioma in South Africa, did some animal studies, correct?
A. Yes.
Q. And they found a total of 11 mesotheliomas occurring, 4 of which were with crocidolite and 4 of which were with Canadian chrysotile, correct?
A. Yes.
Q. In fact, they went on to say, There was no evidence of either less carcinogenicity or less asbestosis in the groups exposed to chrysotile than those exposed to the amphiboles, even though the amounts of dust in the lungs were so different. In particular, the UICC Canadian chrysotile produced as many mesotheliomata as the UICC crocidolite. That's what they concluded.
A. Yeah, and I think one of the points I made in my direct is the difference between the Canadian samples and the Rhodesian ones.
Q. Well -- and you in your papers cited to the 1980 Wagner study where he did the comparative effects of three chrysotiles by injection inhalation to say that there was no

chrysotile mesotheliomas there, but there was -- there was also only one crocidolite mesothelioma, correct?
A. I think so. And I think I'm going to refer to my report, if you don't mind.
Q. That's the only point I wanted to make about that.
You're familiar with the Davis 1978 study, "Mass and number of fibers and the pathogenesis of asbestos-related lung disease in rats"?
A. Yes.
Q. And there they found that there was one peritoneal mesothelioma in 42 animals exposed to 2 micrograms of chrysotile and there was 1 pleural mesothelioma in the crocidolite animals who actually had 5 micrograms of exposure, correct?
A. The important point, though, that I footnoted in my report was that the Davis study found no mesotheliomas in those exposed to the UICC-A samples. And so that's the samples without tremolite. So I think actually that supports my opinion about relative potency.
Q. Well, another study was done by Davis in 1988, "Comparisons of the pathogenicity of long and short fibers of chrysotile asbestos in rats." You're familiar with that study, correct?
A. I think I've seen it at some time, yes.
Q. And what they found was for the long chrysotile, they

found two pleural mesotheliomas and a peritoneal mesothelioma. For the short chrysotile they found one peritoneal mesothelioma, and for the controls they didn't find any mesotheliomas, correct?

A. Again, I would have to look at the exact samples they were using before I could draw any conclusion from it.

Q. Now, in your report you cited the baboon studies that were done by Goldstein and by Hiroshima, correct?

A. Yes.

Q. Now, what you didn't say in your report is in the Goldstein study, they didn't know how much exposure to chrysotile A the baboon was given. So it was hard to compare the fact that no mesotheliomas developed in that animal because we didn't know how much he had compared to what they got from the amosite and the crocidolite, correct?

A. Right, and I think that actually speaks to the fact that dose is important.

Q. And then for the second baboon study in Hiroshima, the reason why there was no chrysotile mesotheliomas is because he had 8.5 to 24 months of exposure, whereas the amosite one had 49 months of exposure which was almost twice as long. And the crocidolite one had 35 months of exposure which was another half as long as the chrysotile. You didn't mention that in your report, correct?

A. Well, I think the point of referencing that all together

was to demonstrate that it's very difficult to cause mesothelioma from chrysotile exposures in animals, and I think that was a perfect example of it.

Q. Well, it might have been less difficult if they gave that animal as much asbestos exposure as they gave the amosite and the crocidolite one, correct?

A. That's not what the study did, though.

Q. Right, and so you don't know whether, in fact --

A. Well --

Q. -- they had given the chrysotile baboon as much exposure as they gave the one for amphibole, that maybe a mesothelioma would have occurred, correct?

A. Well, I think we can make all sorts of guesses about what might have happened. However, they did give the animal chrysotile and no tumors were developing.

Q. But the important point is in your report you didn't mention the fact that the reason why that one of the reasons that might explain why the mesothelioma didn't occur is because the chrysotile baboon got a lot less exposure than the amosite or crocidolite baboon. You didn't mention that, correct?

A. Because we're not doing causal associations based on animal studies I didn't find that it was important to go through every single detail of the animal studies other than to say and to make the point that there is a biologic

rationale for the differences in fiber potency.
Q. So the only thing you thought was important about these baboon studies was to mention that there was no mesotheliomas but not the facts surrounding why there was no mesotheliomas.
A. No, I think the important part in mentioning that was to make sure that there was an understanding that the fiber potency differences are real and that it's much more difficult to make a mesothelioma in a rat with chrysotile than it is with amphiboles.
Q. But you can't make potency determinations if you're not giving the same level of exposure to the representative animals, correct?
A. Well, what would be wrong is if I took that information from an animal study and applied it to a human being, which I wasn't doing. And so if I was making potency assessments in a human being based on that study, I would agree wholeheartedly with you. But that wasn't the point of what I was doing.
Q. Well, but even making potency determinations in animals, it's not appropriate to say that an animal that got less exposure than other animals, therefore they can't get mesothelioma. Anyway, I'm beating a dead horse.
In vitro studies. You would agree that in vitro studies have shown that pure chrysotile -- and they don't put contaminated chrysotile on this level, correct?
A. Right.

Q. When we're doing in vitro studies, we're doing cellular reactions and we're very careful that we're using the fiber, correct?
A. Yes.
Q. And you would agree that it can cause damage to DNA.
A. Like a lot of things. Either spontaneous or exposure related, yes.
Q. But you would agree that in vitro studies have shown that chrysotile can produce a mutagenic event.
A. Yes.
Q. And you agree that chrysotile fibers can cause actual DNA strand breakage.
A. Yes.
Q. Now, you talked about the location of the pleura and you agree that that location is important when we're talking about mesothelioma cases because the asbestos fibers that are translocated into mesothelial tissues play an important role for the induction of an asbestos related serosal disease, correct?
A. Yes.
Q. And you agree it is only the fibers that get to the pleura that are going to cause a mesothelioma, correct?
A. Yes. That's our current understanding of it.
Q. Sure. And so the fact that amphiboles have this long half life inside the lung, all the ones that are staying in

the lung have nothing to do with the development of a mesothelioma in another body part, correct?
A. Well, I think what it does is it speaks to the difficulty with which the lung has in handling the amphibole fiber.
Q. But you agree with the proposition, and you've said it, that the half lifes for crocidolite and amosite are decades.
A. Uh-huh.
Q. Correct?
A. Yes.
Q. And the fibers that are stuck in that parenchyma tissue for those decades aren't going to cause a mesothelioma in the pleura.
A. I think you might be missing the scientific point. The scientific point of looking for fibers in the lung is not only to inform about biopersistence, but it's also a way to make a determination what an individual is exposed to. And the reason that we do that is because you can't readily -- and there's no standards for accessing pleural tissue to do that with. So we just have to be careful that we're talking about lung tissue studies in one context and we can't necessarily apply that to pleural tissue studies which really haven't been done in large measure.
Q. But from a purely physiological standpoint, you would agree with me, if an asbestos fiber is stuck in the lung parenchyma, it has no causal relationship to a tumor that is

arising in the pleura.
A. Not that amphibole fiber.
Q. Correct?
A. But it might have had friends and so that's the part that I'm...
Q. Right, you're using as a surrogate for what other exposures may have been. And the reason why we can't do that with chrysotile is because chrysotile doesn't remain in the lung for any length of time.
A. Well, that's not true. That's not true either. There's chrysotile in the lung of occupationally exposed individuals, non-occupationally exposed individuals. And it's not scientifically accurate to say that all of the chrysotile is removed from the lung and therefore if we can't find it, it must not have existed.
Q. But it's also scientifically true that the half life of chrysotile in lung tissue borders on the range of months rather than years.
A. It depends on the study, but I would accept it's much shorter than an amphibole.
Q. Much, much shorter than amphiboles.
A. Yes.
Q. And where it's going, some of it is getting eaten up by macrophages. Some of it is getting into the lymphatic system and getting washed away. And some of it is getting into the

lymphatic system and sticking in the pleura, correct?
A. I'll agree with all of that.
Q. And the reason why you will agree with all that is because there are scientists who looked at that issue, correct?
A. Yes.
Q. And you're aware of that.
A. Yes.
Q. One of them is Sebastien.
A. Yes.
Q. He did asbestos retention in human respiratory tissues, comparative measures in lung parenchyma and in the parietal pleura and what he found was in the cases studied, the proportion of amphibole type fibers within the lung range from 0 to 100 percent with a mean of 56. And on the other hand, when a pleural sample was positive for asbestos, almost all of the fibers encountered were of the chrysotile type. That's what he found, correct?
A. That's what he wrote. Again, I don't -- I don't put a lot of emphasis on that to determine causation, but that's what he found pathologically.
Q. He said, This study has demonstrated that the retention of asbestos dusts in the parietal pleura was related to type and size. Most of the fibers were short chrysotile fibers.
And he's not the only researcher who found that.

A. No, actually I quite agree with that mainly because those of us who are not occupationally exposed and, in fact, those who are occupationally exposed most of the chrysotile fiber that's found in the lung, and I would think therefore the pleura, is of the short fiber type and that's a clearance mechanism that I think is not injurious.
Q. But not all of the chrysotile fibers that get to the pleura are short.
A. No, I wouldn't say all, but I would say the majority.
Q. Now, Dr. Suzuki, who is at Mt. Sinai, one of Dr. Selikoff's colleagues, he did a study on "Short, thin asbestos fibers contribute to the development of human malignant mesothelioma: Pathologic evidence." And what he found was a disproportion of the type and number of asbestos fibers between the lung and the mesothelial tissues was frequently seen in the malignant mesothelioma cases. Asbestos fibers in the lung were amphiboles or an add mixture of types, chrysotile and amphiboles, where amphiboles were the majority while those seen in the mesothelial tissues were primarily chrysotile. And then he cites at least seven studies that support that proposition, correct?
A. Yeah. The only issue I have with this kind of analysis is that while there's some information about lung tissue burden, there is very little about how much asbestos fibers should be in the pleura in people that aren't exposed, are

exposed, have mesothelioma, don't have mesothelioma. So I'm not really sure what he's comparing the amount of fiber in the pleura to.

Q. Well, you know in Dr. Suzuki's studies, he did have a control population that he compared them to.

A. Yeah, the control population that he has, though, wasn't -- it wasn't clear really who those individuals were and how he was controlling for asbestos exposure.

Q. Now, he says that it was suggested that chrysotile fiber's strong capacity to translocate from the lung into the pleura and/or peritoneal tissue caused the disproportion of the number and types of asbestos fibers between the two tissues.

And those tissues, the pleura and the peritoneum, they are basically the same -- composed of the same type of cells, correct?

A. They are.

Q. It's all one serosal layer?

A. Yes.

Q. When we're born embryonically the pericardium, the pleura, the peritoneum are all really one organism.

A. That's right.

Q. Okay. And you're familiar with this article, "The ticking time bomb of asbestos: It's insidious role in development of malignant mesothelioma."

A. I've read that at some time, yeah.
Q. It says, The usefulness of lung fiber analysis, however, was again called into question in a study comparing fiber measurements between lung parenchyma and the parietal pleura. The authors revealed evidence of predominantly short chrysotile fibers within the pleura, plaques and pleural fibrotic tissue, despite predominantly amphibolic asbestos being noted in the lung. This preferential localization to the pleura casts significant doubt on the accuracy of lung fiber analysis in assessing exposure in patients with asbestos-relate diseases.
That's what they wrote, correct?
A. That's what they wrote.
Q. And what they meant is you can't really do a lung fiber analysis and say aha, there's very little chrysotile here; therefore, this individual who we are examining his lungs 30 or 40 years later was not exposed to chrysotile.
A. Well, I would say it differently. I think what you can't do is do a lung tissue analysis and make causal associations with it. And I think that there's a tendency to do that, and I don't think that that's the right way to do it.
Q. So all this lung fiber burden analysis that the judge heard, that's really not proper evidence for determining causation, correct?
A. It depends on how you use it. I think that what can't be

gleaned from that kind of information is fiber type differences. And so finding amphibole fibers in the lung, for instance, particularly of a certain type, would indicate that somebody was occupationally exposed to them. Finding a certain amount of chrysotile in the lung, on the other hand, whether it's a lot or a little bit, it doesn't help you a lot.

Q. Let's talk about the historical research that you mentioned in your direct. First I want to talk to you about Dr. Wagner's article. You said in 1960 Dr. Wagner found a series of cases in South Africa where not only miners but people living around the mine got mesothelioma and from that evidence he determined that crocidolite was a cause of mesothelioma. Is that an accurate summary?

A. Not exactly that he considered it a cause. He certainly -- again, to use the language properly, found that there was a case series that suggested that further research be done.

Q. And what Dr. Wagner did, as any good researcher would do when they find this phenomenon, is he goes and asks his buddies and say, hey, has anybody else come across this. And what they found was that in 1952, 8 years earlier, Cartier mentioned two cases of diffuse mesothelioma from a Canadian chrysotile mine. A further three cases were described by Van der Schoot in the Netherlands. Unfortunately, no indication is given in the literature regarding the type of asbestos to

which the majority of recorded cases of carcinoma were exposed. However, discussion with management and medical officers of two of the factories in which the majority of the cases reported in Britain were employed, suggests that most of these workers were handling chrysotile asbestos. Now, the possibility that some of these people may have also been exposed to crocidolite dust cannot be excluded.

What this tells us is that in 1960 Dr. Wagner was aware that crocidolite wasn't the only substance suspected of causing mesothelioma, correct?

A. I'm not sure that's what he's saying. I think that instead what he's saying is that some of those individuals who went on to develop mesothelioma handled chrysotile asbestos. I think that's a fairly noncontroversial point.

What he wasn't able to do, and you can't with two cases, is try to determine what else those individuals were exposed to.

Q. Well, let's talk about Dr. Selikoff in 1965. So five years later Dr. Selikoff says, look, if the hypothesis that mesothelioma associated with asbestos contact is a special case of asbestos cancer caused by exposure to one kind of asbestos fiber, crocidolite, it's correct the problem is a rather limited one. Although it would be important in the areas in which crocidolite is mined, and to those working with it, the United States asbestos industry could be advised to

avoid this type of fiber in favor of other forms of asbestos.

So what Dr. Selikoff decided to do is test the hypothesis. Our investigations could serve to test this hypothesis. Crocidolite has had a small role in the United States asbestos industry and that -- but recently neoplasms associated with asbestos exposure generally occur only 20 to 40 years after exposure. If mesothelioma could be found with increased frequency in association with asbestos in this country, it would demonstrate that this tumor was another neoplastic hazard of asbestos exposure in general and not limited to one area or to one type of fiber.

That's what he wrote in 1965, right?

A. Yes.

Q. And in that very same publication he reported the fact that in their study of 307 consecutive deaths among asbestos-insulation workers -- now, those insulation workers weren't exposed to very much crocidolite at all, correct?

A. That's right.

Q. They were exposed to 90 percent chrysotile and maybe 10 percent amosite, correct?

A. Well, I don't know the exact proportion and I don't know the percentage of crocidolite in any of these --

Q. But it was predominantly a chrysotile exposed population.

A. Yeah, I think that's right.

Q. Okay. "They found ten deaths caused by mesothelioma of

the pleura (four cases) or peritoneum (six cases). This is an extraordinarily high incidence for a tumor generally so rare that its not separately coded.

"It appears mesothelioma must be added to the neoplastic risks of asbestos inhalation, and joins lung cancer (53 of 307 deaths) and probably cancer of the stomach and colon (34 of the 307 deaths) as a significant complication of such industrial exposure in the United States."

That's what he wrote, correct?

A. And I think this is exactly the reason why I wanted to be sure to present as comprehensive a picture as possible because to say that somebody was just exposed to chrysotile and therefore that causes mesothelioma is not going through the proper scientific method. And so while some of these people were certainly exposed to chrysotile. The fact that they were also exposed to amphibole asbestos and putting that in the context of all the medical literature available makes it very difficult to -- for me to say that chrysotile elevates the risk.

Q. But at this point in time, what you're saying is if I find any amphibole, that must be the cause and you're just ignoring the fact that this population had -- their majority exposure was to chrysotile.

A. I'm not ignoring the fact at all. As a matter of fact, I think that as a lot of my comments in the direct would

indicate, I'm taking into account all the opportunities to study both fiber types and make that determination. So that includes studying brake workers. That includes gas mask manufacturers. That includes mining communities in South Africa. So if we're trying to get to an honest answer about risk, we have to include all of that literature.

Q. Let's keep on looking at the literature. So in 1966, a year later, Ward O'Donnell, Richard Mann, and John Grosh wrote their paper, "Asbestos, an extrinsic factor in the pathogenesis of bronchogenic carcinoma and mesothelioma" where they report on 55 asbestos textile workers who had pathologically proven asbestosis and 28 malignant neoplasms were found, 23 bronchogenic carcinomas, and 5 mesothelioma of the peritoneum and the pleura. Now this is 1966.

You're familiar with this article, correct?

A. Yes.

Q. And what they said was, "The plant involved in this study used the chrysotile type of asbestos fiber almost exclusively. The neoplastic hazard results from exposure to asbestos in general rather than to any one particular fiber type."

That was what their conclusion was in 1966 in an historical perspective.

A. Yeah, in 1966. And I think the comment almost exclusively is revealing because, again, we don't know in that particular cohort if there was a mixed exposure setting or

not. And I think that that's the difficulty in going back to a study from 1966 and trying to draw conclusions about it.
Q. That's true about every cohort we're going to talk about today is they're mixed exposure cohorts to a certain extent because you just said in the beginning of this examination that you don't believe that there's anybody in the world that's exposed exclusively to pure chrysotile fibers.
A. Well, I didn't say that. And --
Q. But would you agree that that's --
A. No. No. I wouldn't agree with that. There is evidence that I presented earlier today that gives you the best opportunity to look at chrysotile exposures. And so whether or not there's a medically significant amount of tremolite associated with those or not -- you've got the South African mining population. You've seen the difference in the New Orleans asbestos in those plants. You've seen the gas mask manufacturers. You've got the biologic rationale for why the fiber potency differences exist. And that's why it's very important to look for those opportunities to study the different fiber type rather than draw a conclusion with one particular study or the other.
Q. Now, you know Dr. Lemen is a Ph.D epidemiologist, correct?
A. Yes.
Q. Former assistant surgeon general of the United States

Public Health Service. He wrote a paper on chrysotile asbestos as a cause of mesothelioma where he applied the Bradford Hill causation criteria. And you're familiar with those criteria, correct?

A. Yes.

Q. And he said that "this paper examines one proposed model for establishing causation as presented by Sir Austin Bradford Hill in 1965. Many policymakers have relied upon this model in forming public health policy as well as deciding litigation issues."

And it's a very well accepted criteria to look at to determine cause and effect, would you agree with that?

A. It is with an important caveat. That there has been a statistically significant excess risk demonstrated with that particular exposure.

So the first thing you need to do if you're going to apply the Bradford Hill criteria is show a statistically significant increased risk and then make the causal association assessment. Just by showing the statistical increased risk, you're not showing that something causes a disease.

Q. But wasn't Sir Bradford Hill's entire point was not to be a slave to statistical significance; that we have to look at all of the factors. We have to look at biologic plausibility. We have to look at coherence. We have to look at animal

studies. We have to look at all of the factors.
A. Couldn't agree more. Couldn't agree more.
Q. Now, what Dr. Lemen said is "chrysotile asbestos meets Hill's nine proposed criteria, establishing chrysotile asbestos as a cause of mesothelioma."
And he explains what we're doing right now. And what we're doing right now is there is a debate. Correct? There is a debate in the world science as it applies to human exposure to pure chrysotile-containing products.
He says, "It's academic at best as there appear to be few if any pure chrysotile deposits unequivocally identified or reported in the scientific literature; nor has any product purported to contain only chrysotile been conclusively shown to contain uncontaminated pure chrysotile."
And you would agree with that, right? That was your sentence at the beginning of this examination, basically.
A. No. My comment about the Lemen paper, and I know that there's going to be other experts that have offered an opinion on this specifically about the Lemen paper so I won't belabor the point. But there is an occupationally exposed cohort that was exposed to low dose chrysotile whether tremolite contaminated or not that showed no excess risk for development of the disease. And in this paper there was no mention of those many cohort studies in the auto mechanics.
Q. Well, he wrote a separate paper on the fact of causation

of asbestos in auto mechanics. You're aware of that.
A. I am. And I don't know how one can reach the conclusion if you review all of those epidemiologic studies that working with friction materials, brake work, elevates the risk.
Q. But he's a Ph.D epidemiologist. He reviewed the evidence and in his opinion he believes that, in fact, there is sufficient evidence, even though the epidemiology is equivocal, that asbestos in brakes cause mesothelioma.
A. That's his opinion. I don't agree that the epidemiology is equivocal
Q. I understand that, but that's what I'm saying. It's a debate, correct?
A. I think that's a proper classification of it.
Q. In fact, he goes on to say, "If and when such deposits or products are identified," these pure ones, "the fact remains that chrysotile alone can cause mesothelioma, as demonstrated in this paper, when fitting the existing scientific knowledge into the parameters of the Hill causation model. The exact potency of chrysotile, per dose, needed to cause mesothelioma, when compared with the amphiboles, remains controversial and has been discussed else where. However, even when potency on a dose-by-dose basis is considered, the fact remains that chrysotile is capable of causing mesothelioma and that no safe dose has been identified below which a risk of developing mesothelioma no longer exists.

That's his opinion, correct?
A. Yes.
Q. He published that opinion.
A. Yes.
Q. And there are others who agree with that opinion.
A. I'm sure there are.
Q. And in fact, I want to talk to you about this Acheson study. This is the study that you referred to on your direct, correct?
A. Yes.
Q. Mortality of two groups of women who manufactured gas masks for chrysotile and crocidolite asbestos, a 40-year follow up. You said that the difference between the two groups of workers was one was with chrysotile and one was with crocidolite, but there were some other differences, correct?
For example, the population that was manufacturing the chrysotile gas masks began in 1936 and it ended when the war ended.
The population of workers that were making the crocidolite gas masks started in 1927 and made them until 1969.
So we have a significantly longer exposure period between the chrysotile gas mask workers and the crocidolite gas mask workers, correct?
A. I think that's right, but the point I think is is that

it's long enough to -- for latency period to run so that you would see disease if it was present.
Q. There was also another difference. The difference was the chrysotile process was mechanized, which meant it was done in a factory. And factories in England in the 1940s, they already knew about asbestos through Merriwether and Price and so they were instituting industrial hygiene controls to control exposure, correct?
A. I don't know.
Q. But the crocidolite gas masks were made by hand and so there would have been more exposure if you're hand making something than when it is being mechanized.
A. I don't know about that.
Q. Well, there was mesothelioma in the chrysotile population, correct?
A. Yes.
Q. And they recognized that maybe they had reason to believe that that person might have done some work in another Blackburn factory that had crocidolite, but they don't actually give us any data to support that reason to believe, correct?
A. Correct.
Q. Let's talk about the Canadians. You talked on direct about the fact that there is more tremolite in one area of Canada than in another. That information was all taken in

consideration by the IARC when they did their 2012 evaluation, correct?

A. I assume so.

Q. In fact, they said, "The fact that chrysotile asbestos mined in Quebec is contaminated with a small percentage of amphibole asbestos has complicated the interpretation of these findings. McDonald found in a case control study for mesothelioma in the Thetford Mines in Quebec that an association with asbestos exposure was evident in the mines from a region with a higher concentration of tremolite and not in another region with lower concentrations of tremolite."

They go on to say, however -- and I never know how to pronounce this in French. Begin?

A. Begin.

Q. "Noted that although tremolite levels may be 7.5 times higher in Thetford than in asbestos, the incidence of mesothelioma in these two Quebec mining towns was proportional to the size of their work force. This suggests that the tremolitic content of the ores may not be a determinant of mesothelioma risk in Quebec. Separate analyses for workers at the Thetford and asbestos mines and mills did not demonstrate a different exposure response relationship for asbestos and mesothelioma in these two mining areas."

That's how IARC interpreted the discussion that you did on direct, correct?

A. I think that's how they interpreted. There's a lot of speculation in that comment.

Q. Let's talk about South Africa. They say in a mesothelioma case control study in South Africa, an association was reported with exposures to crocidolite and amosite asbestos, but no cases were found to have been exclusively exposed to chrysotile asbestos. One possible explanation for these negative findings for chrysotile is that South African chrysotile asbestos may contain relatively little tremolite. Another possible explanation is that chrysotile mining began later."

They didn't begin mining chrysotile in South Africa any the mid 1960s, correct?

A. Yes.

Q. And the production levels were lower. They mined more crocidolite and more amosite than they did chrysotile.

And in fact the work force was smaller. There were less chrysotile miners than there were amphibole miners, correct?

A. That's correct.

Q. And in fact, most of the chrysotile miners were African Africans.

A. Yes.

Q. They were blacks.

A. Yes.

Q. And there has been very little documentation kept about

what happened to that work force as it progressed --
A. Well, I think importantly, though, Rees followed up this study in 2001 with lung tissue fiber analysis that did find that there was more risk in the individuals that had amphibole in their lungs.
Q. But cases of mesothelioma have been reported among asbestos miners in Zimbabwe which has been reported to be uncontaminated with tremolite asbestos. You're aware of that.
A. This is the article which subsequently found anthophyllite in that cohort in 1996.
Q. You're aware of Margaret Becklake, correct?
A. Yes.
Q. She wrote an article in 2007 and she found that in Zimbabwe between the two chrysotile mines of Shibanie and Gaths, they found 36 cases of mesothelioma that were identified.
A. Yes, I'm aware of that study.
Q. Okay. Now, there are authors, and I know you disagree with this, but Allan Smith is another epidemiologist, correct?
A. Yes.
Q. He's from California.
A. Correct.
Q. You've been in trials where he's been on the other side, correct?
A. I won't hold that against him, the California part.

Q. But he's a Ph.D.
A. I think so, yes.
Q. He wrote a peer reviewed article.
A. Correct.
Q. His conclusion based on his review of the evidence is that chrysotile asbestos is the main cause of pleural mesothelioma, correct?
A. That's what he said.
Q. Okay. And he -- his conclusion is there's three points. One, chrysotile asbestos is a potent cause of pleural mesothelioma; two, the large majority of mesothelioma is attributable to asbestos exposure; and three, chrysotile asbestos has been the major fiber type used.

"Based on this evidence, we conclude that chrysotile asbestos is by far the main contributor to pleural mesothelioma causation in the U.S. and other countries in which it has been the predominant fiber type."

A. I understand that pulling these snippets out is the way things are done, but you have to know how and why he reached that conclusion.

The how he reached that conclusion is to consider all of the cohorts that he studied, chrysotile cohorts, quote unquote. Those were clearly mixed cohorts that he's studying.

You also have to understand the point that he excluded all of the cohorts that showed no elevation of risk, i.e., in

the auto mechanics studies that simply had no excess risk associated with it.

So I would have to wonder why exclude the studies that showed no excess risk and why label the studies that he did include as chrysotile-only studies when they were clearly mixed studies.

Q. But, of course, Dr. Smith is not by himself. There are other researchers that have reached similar conclusions. Dr. Landrigan, Nicholson, Suzuki, and Landou from Mt. Sinai, they said that clinical and epidemiologic studies have established beyond all reasonable doubt that chrysotile asbestos causes cancer of the lung, malignant mesothelioma of the pleura and peritoneum, cancer of the larynx, and certain gastrointestinal cancers. Chrysotile also causes asbestosis, a progressive fibrotic disease of the lung. The risk of these diseases increases with cumulative exposure to chrysotile and also with time since first exposure.

Now, you disagree with that, but that's what those researchers found, correct?

A. They do.

Q. You know Leslie Stayner, correct?

A. I know who Dr. Stayner is.

Q. Another Ph.D.

A. Yeah, another Ph.D. I haven't met him.

Q. He reviewed this issue. He called it the "amphibole

hypothesis." There is a group of researchers like yourself who support the hypothesis that it's not the chrysotile causing the disease, it's the contaminant tremolite. And there's another group of scientists who say, no, we think it's all of it, both the chrysotile and the tremolite, true?

A. That's the debate.

Q. And he says, "Our view of both the toxicologic and epidemiologic literature strongly supports the view that occupational exposure to chrysotile asbestos is associated with increased risk of both lung cancer and mesothelioma. The hypothesis that these observations may be attributable to trace amounts, less than 1 percent of tremolite contamination may seem to be primarily of an academic interest because chrysotile exposures in workers and in the public are also contaminated with tremolite."

That's his view, correct?

A. That's his view.

Q. There was a cohort study, a meta analysis that was done in China where they concluded there are excessive risks of lung cancer and mesothelioma among workers exposed to chrysotile fiber alone, and likely no convincing indication of an etiological association between chrysotile exposure and cancers at other sites."

You're familiar with the Lee paper, correct?

A. I am.

Q. That went backwards.

They started out, they said, "There's been a heated debate on the carcinogenic effects of exposure only to chrysotile during the recent 20 years."

Been going on for two decades, correct?

A. Or more, yes.

Q. Every trial that you've been in, we've had this one side/other side presenting their views of the evidence.

A. That's right.

Q. And you don't believe -- it's not your belief, is it, that somebody like Dr. Smith or somebody like Dr. Lemen or somebody like Dr. Stayner is somehow fraudulent because they have a different view of the same scientific papers that you do?

A. No.

Q. It's a good faith academic exercise, correct?

A. I assume so. I don't know what an individual's intention is, but I assume so.

Q. And given the extent of that debate, that's why we have juries because they are the ones who evaluate what you say, they evaluate what Dr. Lemen says, and they come to their conclusion on who they believe has the stronger point of view.

A. That's how it works.

Q. So what these guys did -- and again, that amphibole hypothesis postulated that amphiboles are the major cause of

mesotheliomas in asbestos workers. The lung burden of chrysotile and non-asbestos fibers bear no relation to the occurrence of mesotheliomas and lung cancers and that amphiboles are more potent than chrysotile in inducing fibrotic lung disease and associated lung cancer.

That's a summary of what the amphibole hypothesis is, correct?

A. Correct.

Q. What they did to test that hypothesis -- well, thus a controversy apparently existed. They even named the two warring factions. Those that support the amphibole hypothesis are called the chrysophiles and those that opposed it are the chrysophobes. And in fact, there was a researcher in England who actually wrote a paper: "Chrysophiles versus chrysophobes." You've seen that?

A. No.

Q. The column?

A. No.

Q. I'll give it to you sometime.

A. Thanks.

Q. Our final literature search identified 25 articles reporting 26 cohorts that met their inclusion criteria. And they identified they were from China, USA, UK, Canada, Italy, Sweden, Denmark.

They found that chrysotile constitutes 95 percent. They

also found no threshold. And they concluded that chrysotile alone has excessive risks of mesothelioma. A conclusion you disagree with, correct?

A. And the way the paper was done methodologically, yes.

Q. One person you do agree with is Charles Yarborough who did a paper on "Chrysotile as a cause of mesothelioma." And he, like you, is a litigation expert who comes into court on behalf of companies to argue the chrysophiles' point of view, correct?

A. I suppose that's a fair and accurate...

Q. Let's talk about what level of exposure to asbestos doesn't cause mesothelioma.

You're aware of the British Thoracic Society?

A. Yes.

Q. In fact, you're a pulmonary doctor yourself, are you not?

A. I am.

Q. And they come to the conclusion based on their review of the literature that there is no evidence for a threshold dose of asbestos below which there is no risk. They would disagree with you when you say, hey, you can be exposed to tons of chrysotile and it's not going to cause any problem. That's not their conclusion, correct?

A. And not exactly what I said. I think that when you look at statements such as this, there is a difference between saying there is no evidence for a threshold dose and the other

statement that would say that there is no known threshold. Those are two very different things. I think that a lot of times phrases like this get written and it leaves one with the impression that there is no threshold, and that's wrong.

I think the issue is is whether or not you can define precisely where that threshold exists and that's a separate scientific matter.

Q. And for 20 years, thousands of publications, no scientist from the peer reviewed literature has been able to convince everybody that there is some measurable amount of asbestos that below that number you're not going to get mesothelioma. That's why these statements exist, correct?

A. Well, I think they exist because the studies that have been published aren't set up to define the threshold. They're not setting out with the intention that I'm going to find the minimum amount of dose below which no risk exists. That's not how the studies are set up. And so that question really won't ever be answered because we're not going to be able to design a study that gives you that kind of precision.

All you can do is use what's out there in the medical literature to make the comment that I believe in which is is that there is a threshold. We just don't know exactly the exact fiber CCs year that it is defined by.

Q. But certainly if it was true that the threshold was you can be exposed to a hundred fibers per CC of chrysotile and

not get mesothelioma, if it was that obvious, there would be people that would agree with that and publish that, correct?
A. Not necessarily. I think that the point really is is that the studies aren't set up to show where there is no risk. The studies are set up to show where there is risk. And that's been in hundreds of fiber years in the studies that I mentioned during my direct with regards to chrysotile mining. So that's the information that you have.
Now, as I mentioned, drawing a straight line from those kind of exposures back to zero makes no scientific sense to me.
Q. There are other researchers out there who have tried to determine how low you can go, including Iwatsubo in France and Rodelsperger in Germany which admittedly are mixed dust exposures, but, again, even as mixed dust exposures, they're predominantly chrysotile exposures, and they found that levels lower than what they were regulating asbestos exposure at was causing significant levels of mesothelioma, correct?
A. Well, as they certainly concede in their papers, they didn't really have a clear understanding about what the dose was at these very low levels. And in fact, put their exposure estimates in quotations to signal to the reader that they weren't exexactly sure what kind of low exposures we're talking about.
Q. It was a panel of five certified industrial hygienists

experts who had to retrospectively construct the exposure because obviously nobody was taking measurements at the time of the exposure so that's the best we can do, correct?

A. I understand that was the best they could do. I'm just saying that it wasn't good enough to reach the conclusions that I think you're suggesting.

Q. Now, the British Thoracic Society recognizes that there has been much debate about the etiologic role about the chrysotile white asbestos. However, a recent World Health Organization review has concluded that chrysotile asbestos does indeed pose an increased risk of mesothelioma in a dose-dependent manner. This form of asbestos is also the most widely used.

And dose-dependent means, like those response curves, more exposure greater risk.

A. Right.

Q. And we'll talk about that in a little bit more detail.

You're familiar with Gunter Hillerdal?

A. Yes.

Q. Very well respected researcher out of Scandinavia.

A. Yes.

Q. He wrote a paper on "Mesothelioma: Cases associated with non-occupational and low dose exposures." His conclusion, "There is no evidence of a threshold level below which there is no risk of mesothelioma."

He goes on to say, "There is no proof of a threshold value - that is, a minimal lower limit below which asbestos fibers cannot cause the tumor - and thus it is plausible that even such low exposure can cause mesothelioma (even if the risk is extremely low)."

And when we talk about a dose-dependent disease, obviously the lower the exposure, the lower the risk; the higher exposure, the higher the probability you're going to get the disease, correct?

A. Right.

Q. Okay. You're familiar with the "Environmental Health Criteria 203" from the World Health Organization and others?

A. No.

Q. You've never seen this book?

A. I don't think so.

Q. Little red book about 500 pages?

A. I may have seen it. I certainly don't recall what's in it.

Q. I'll skip it. I'll skip it.

Here is the World Health Organization which the British Thoracic Society was referring to. And they have reached the conclusion that there's no threshold that has been identified for carcinogenic risk of chrysotile. Bearing in mind that there is no evidence for a threshold for the carcinogenic effect of asbestos. And that increased cancer risks have been

observed in populations exposed to very low levels. The most efficient way to eliminate asbestos-related disease is to stop using all types of asbestos.

They don't advocate the continued use of chrysotile, correct?

A. That's what it looks like.

Q. You're familiar with the Helsinki criteria.

A. Yes.

Q. Bunch of well-respected experts --

A. Yes.

Q. -- got together. And their position is, "Mesothelioma can occur in cases with low asbestos exposure. However, very low background environmental exposures carry only an extremely low risk." Just what we've talked with. Lower the dose, lower the risk; higher the dose, higher the risk.

And they say, "An occupational history of brief or low level exposure should be considered sufficient for mesothelioma to be designated as occupationally related."

In your practice that's how you make the connection, correct, is an exposure history that you elicit from your patient?

A. Yes.

Q. That's the sole basis in a lot of cases to say that mesothelioma is asbestos-related or not, the only difference between you're looking solely for exposure to amphibole

asbestos.
A. Yes.
Q. These guys say any exposure. That's the true difference between your opinion and theirs, correct?
A. Looks to be.
Q. Okay. And you're familiar with Laura Welch and her paper, and this is about brake mechanics. And these 51 scientists disagree with you. They agree that, in fact, there is sufficient evidence to say that exposure to asbestos from brakes can cause mesothelioma, correct?
A. That's what they say.
Q. Okay. They also say -- and this is just to show that they have lots of experience, these 51 people. They're not just Joe Blow off the street. These are epidemiologists and scientists and toxicologists, correct?
A. Correct.
Q. Very well credentialed, great institutions.
A. I think so.
Q. The scientific community is in the consensus that even brief and low level exposure to asbestos can cause mesothelioma. The main stream scientific community has long recognized and continues to recognize today that there is no safe level of exposure to asbestos.
And they go on to note that NIOSH, the National Institute of Occupational Safety and Health, has said that cancer risks

have been demonstrated at all fiber concentrations studied to date. Evaluation of all available human data provides no evidence for a threshold or safe level of asbestos exposure.

That's what those 51 people have concluded, correct?

A. Yes.

Q. Okay. Attempts to postulate thresholds for exposure have been dismissed as logical nonsense. The lack of a defined safe level for exposure to asbestos has been supported by subsequent research. For example, a large French study recently concluded that substantial excess mortality occurs at exposure levels below current regulatory levels. And that's that Iwatsubo study, correct?

A. Yes.

Q. And that's I-w-a-t-s-b-u -- a-b-u. I'll get you the spelling.

You recognize that when OSHA put in its standard of 02 fibers per CC back in 1986 they recognized that that wasn't going to prevent mesothelioma cases in some individuals?

A. I think the standards were put in place for non-malignant diseases largely, but...

Q. They were put in for?

A. Non-malignant diseases.

Q. And they recognized that no matter how low we go, we're not going to be able to ensure that some people exposed at that level are not going to get mesothelioma, but that's as

practically as low as we can regulate it.
A. I think that was their general comment. I don't know if they explicitly said that, but I think that was their general opninion at the time.
Q. I think they say that leaves a remaining significant risk. However, as discussed below in an earlier document, OSHA believes that this is the practical lower limit of feasibility for measuring asbestos levels reliably.
In other words, the only way we can go any lower is just to ban asbestos and they were reluctant to do that; is that true?
A. Or develop other ways to test the air.
Q. Back to the ticking time bomb. They agreed that the potency of asbestos fibers to induce mesothelioma may vary to some degree, but there is no safe level established to justify the use of this product.
Let's talk about potency. You agree that there are other potency estimates that you haven't discussed.
A. Yes.
Q. One of them is by Paolo Boffetta?
A. Yes.
Q. He is an epidemiologist?
A. Yes.
Q. World class?
A. Yes.

Q. Very well respected?
A. I think so.
Q. He wrote a paper back in 1998: "The health effects of asbestos exposure in humans, a quantitative assessment." And he said, "Pleura mesothelioma is a malignant neoplasm which is specifically associated with asbestos exposure. The risk is linked with the cubic power of time since first exposure, after allowing for a latency period of ten years." That's the Peto formula.
A. Yes.
Q. "And depends on fiber type as the risk is about three times higher for amphiboles as compared to chrysotile. Environmental exposure to asbestos is also associated with mesothelioma risk."
So based on all the studies that have been published up to 1998, Dr. Boffetta's point of view was there's about a three time differential between the amphiboles and chrysotile.
A. Yeah. I remember reading that and don't know how he reached that conclusion. It's not really well referenced.
Q. This is the chart, though, you used in our trial in October. I noticed you've truncated by taking some out --
A. Yes.
Q. -- so we're down to this chart.
A. Yes.
Q. And what we did is -- this came from the Hodgson and

Darnton.
A. Yes.
Q. And that's where they came up with it.
And what we did in October and I want to do very quickly here is find out how did they get to this ratio of 1 to 100 to 500. In order to make that ratio, they had to have studies that study chrysotile, studies that studied amosite, and studies that studied crocidolite and then compare them, correct?
A. Right.
Q. And you're aware that for the crocidolite numbers, they've been criticized in letters to the editor that they didn't know what they were doing. That they were not using proper industrial hygiene protocols for the crocidolite numbers because they were just guesstimates. And that's actually a word that's in the Hodgson and Darnton paper, correct?
A. I wasn't aware of the criticism. I remember the guesstimates part in their paper, yes.
Q. But for the crocidolite portion, they looked at only four studies. That composed the entire amount of material they looked at to make this analysis, correct?
A. Right.
Q. They looked at Carolina. And the judge has heard something about this Carolina plant in Marshville. That was

one of the four studies they used to come up with this number. Quebec, Balangero, and they used the old one, Piolatto, back in 1990. They used the old Carolina one in 1994. And then they used a Connecticut study by McDonald in 1984.

So when they did this 1 to 100 to 500, they were using studies that are now almost 20 years out of date in some cases.

A. Yes.

Q. Actually, 30 years if we go down to the Connecticut study.

A. Some of those studies were old. The paper was published 13 years ago so...

Q. And you're aware that there have been updates to these cohorts.

A. Yes.

Q. One of these updates the judge has heard about is the Loomis paper in North Carolina. That's where they found three mesotheliomas in plant 3 where they didn't work where any of the amphiboles were and four mesotheliomas in plant 4 where there was no record of amphibole asbestos and it might have been underreported.

Now, you agree that in some of these studies they're using the international code of disease -- or international disease classification. I don't know why they call it ICD if it's IDC.

A. I think it's a French.
Q. It's a classification where if somebody dies of diabetes, you look it up. It's number 106. And then you can put that on the death certificate and everybody knows what they died of, right?
A. Right.
Q. The problem with mesothelioma is it didn't have its own disease classification until ICD code 10.
A. Right.
Q. Which was sometime in the late '90s --
A. I think that's right.
Q. -- early 2000.
A. Yes.
Q. So if somebody died of mesothelioma, they'd either put it down as a cancer of the pleura or maybe a cancer of the abdomen or carcinomatosis or something. But if you're going back, the way they do these mortality studies is they thumb through these death certificates and they have to then code them. And without a pathological evaluation, sometimes it's difficult to tell what the disease is.
A. At times.
Q. And in fact, for mesothelioma there's been plenty of studies that have reported there is an underreporting of mesothelioma because of this coding problem.
A. Actually it's worked both ways, but I think that the

classification point is a valid one.
Q. Okay. You said that you saw these documents that came from the Marshville plant.
A. Yes.
Q. Now -- and I asked Dr. Garabrant this. There were 7,000 of them. Did you do the individual review of those?
A. No.
Q. Basically what happened is Mr. Schachter and Mr. Harris said, hey, we got all these documents. We culled it down. Here's what you should look at, right?
A. Yes.
Q. They didn't go to the UNR trust, did they?
A. I don't know.
Q. You didn't see the statement by Erle Plumber who was in 1957 promoted to the general sales manager --
MR. SCHACHTER: Excuse me, Your Honor, is this an exhibit? May I have the exhibit number, please? What exhibit number is it?
MR. GEORGE: Whatever our next in line is.
MR. SCHACHTER: Well, if it hasn't been produced before --
MR. FINCH: Your Honor, the court's pretrial order said exhibits for purposes of impeachment don't have to be disclosed.
MR. SCHACHTER: Well, I don't know that this is

impeachment. This is --

MR. GEORGE: Well, let's --

MR. SCHACHTER: Trying to put in some hearsay.

MR. GEORGE: Let's just hear what the evidence is.

THE COURT: Overruled. Go ahead.

Q. So he was -- he started out as a sales trainee --

MR. SCHACHTER: Well, objection, Your Honor. It hasn't been offered in evidence yet. And the witness doesn't have any knowledge of it so --

THE COURT: Do you know anything about it?

THE WITNESS: No, sir.

THE COURT: Okay. Let's -- we'll go on to something else.

Q. Well, let me ask you this. If UNARCO had documents where they said that all of the amosite was in their Bloomington plant and that all that was in their Marshville plant was textiles that contained chrysotile, that's not information that your counsel gave you, correct?

A. I didn't get the information.

Q. Okay. You haven't gotten anything out of UNARCO.

A. No.

Q. And you know UNR Industries ran that mill, ran that textile plant from the mid '50s until 1963 when they sold it to Johns-Manville.

A. Yes.

Q. So all you have seen is the Manville side of things from 1963 forward.
A. I brought with me, and I think it's referenced in my report, what exactly I reviewed.
Q. Okay. Well, one of the things that we do know because we do with Dr. Garabrant is that in answers to interrogatories, UNARCO has said Bloomington is the place where unibestos, pipe insulation, blocks, wovenstone, insutape, insutube and insubestos was made.

First of all, let me ask you this. As a pulmonary doctor in your normal practice, would you ever rely on a bunch of corporate documents handed to you by a patient and said this is what my exposure is?
A. It would depend on the setting. Obviously, this isn't in my office seeing a patient so it's a different situation.

The documents that were provided for me that I referenced in my report were given to me, I think, with the idea that the Marshville plant was important in this case. I think it was important in that study. And therefore, I wanted to look at anything that was available to demonstrate that there was amphiboles present in that plant. And so to my satisfaction those documents that were provided me showed that.
Q. But you would agree with me, you're not an expert, are you, in evaluating what the volume of material was and what the type of material it was in the Marshville plant.

A. No. All I can do, though, is just take the documents that were provided for me and I can show you where -- what I relied on in those documents.
Q. And what you have relied on is essentially maybe a hundred pages.
A. I don't know. Maybe, yes.
Q. So whatever else was in that 6,800 pages, be they invoices that show tons of chrysotile being imported to the plant, you don't know what that is because your counsel didn't share those with you, correct?
A. Well, I don't know what they were.
Q. Right. You got a selective hundred pages out of a 7,000 page production.
A. And I made the determination that what I saw was evidence that there was amphiboles in the Marshville plant, so that's sufficed for me.
Q. But you're not a product expert. You don't know where insubestos was made, do you?
A. No. All I can do is read the products and make a determination whether it was likely or not that there was amphiboles in that plant, and I think there was.
Q. Would you agree with me that it's likely in a textile mill that you're going to be using mostly chrysotile asbestos?
A. I don't know what the percentage was. I wouldn't argue with the fact that there was chrysotile in that plant. The

question was was there amphibole as well.
Q. And you don't know what the percentage is because that's not really your area of expertise, is it not?
A. Well, I can certainly understand the numbers if they're provided to me. I don't go out and find the numbers myself.
Q. What I find that's curious is in looking at the bibliography of your report, I don't see the Dement paper where they did an evaluation of the 39,000 air samples, historic air samples they had from 1964 through the 1970s. Is that something that you relied on?
A. I've heard about that paper. I didn't rely on it for this. I think there was an issue about who owned the plant and what was being manufactured there at a certain time frame, and I understand there was a friction plant at one point and a textile plant later.
Q. Would it be important in formulating opinions about whether there was amphibole present in a plant if somebody had taken a transmission electron microscope with the capacity of selected area defraction and evaluated nearly 40,000 air samples and found only 16 that would have been either tremolite or actinolite?
A. Without having seen the study, it would only be important if those samples were taken at the time that the Loomis paper was evaluated.
Q. Well, the samples were taken. They say in the paper the

samples were taken from 1963 through the 1970s and that encompasses the Loomis cohort, correct?
A. Again, I would just have to see the paper. I just don't know the answer to that.
Q. So you would agree that at this point in time, you can't really fully -- you can't give an opinion to a reasonable degree of medical certainty that, in fact, amphibole asbestos was used in the Marshville plant.
A. No, I actually agree with Dr. Brodkin, who I believe is an expert for you, that it's very likely that there was amphiboles there based on the documents I reviewed.
Q. Of course, Dr. Brodkin hasn't reviewed some of these other documents as well.
Let's go forward.
Based on Loomis, based on the fact that there was -- they found an additional mesothelioma from when Hodgson and Darnton did it back in 19, whenever it was, 1994, I believe is what they relied on. Their reference is 1994. At that time there were only two in the study, correct?
A. Yes.
Q. And so when Loomis went back, they got eight, correct? That's what Loomis found?
A. Yes.
Q. And based on that, Hodgson and Darnton, the guys that did the 500 to 100 to 1 wrote a letter to the editor, correct?

A. Yes.
Q. And they said, whoa, thank you. We welcome the appearance of the new analysis of asbestos-related mortality from Loomis which constitutes an important addition to the available evidence.
So this issue about whether there is any amphibole there or not is a fairly significant issue, correct?
A. I think it is. And I think that there is no indication from this letter that Hodgson and Darnton were aware of the possibility that there was amphibole there or not.
And further, I think that the letter was quick to say that based on the follow-up in the Connecticut annual and its cohorts, that their statistical and quantitative analysis was still valid.
Q. Well, we'll see what they said, but let me ask you this. Well, it just flew out of my head what I was going to ask you.
Oh. You haven't seen any evidence in any piece of paper from the debtors' attorneys that shows an invoice that says here is amosite asbestos and it's going to the Marshville plant. You've not seen that.
A. I can show you the information that I have.
Q. I already know what it is because it's in your report.
A. Yeah.
Q. But it's all by inference, correct?
A. Yes, it is. It has to be. I mean, that's what we were

looking for when we were looking for evidence. I mean, this has to be by inference.

Q. And a little supposition as well.

A. I wouldn't say that. I think that there is good evidence that there were amphiboles in that plant.

Q. Good evidence?

A. I think so.

Q. Based on Loomis, these authors said, The risk of mesothelioma derived from these new data is higher by a factor of 10 than what emerged from our meta-analysis, right?

A. Again, they're assuming that Loomis is a non-amphibole cohort in plant 4.

Q. And they're also assuming that there were no mesothelioma cases out of the Connecticut plant.

A. Yes.

Q. And they're assuming there were only four cases out of the Balangero Italian miners, correct?

A. I don't remember how many from Balangero they assumed, but I'll accept that.

Q. Two because it was based on Piolatto. I think I have a slide here.

Now, there are other textile plants where they found lots of mesothelioma cases, correct?

A. Yes.

Q. In fact, here's one where they did friction products and

packing. That plant, 99 percent of what that plant used over its working life was chrysotile. They used upwards of 5 to 6 thousand tons per year of chrysotile. They used 375 tons of amosite for three years. And they used 7,500 pounds of crocidolite once. So that's a 99 percent chrysotile-exposed population. And they found 17 mesothelioma cases out of that group, correct?

A. I don't know. You flashed rather quickly by the front of the article.

Q. Oh, it's the Wagoner, Robinson, Lemen. You're not familiar with that paper?

A. I don't think so, no.

Q. Okay. Would it be your opinion based on those facts that none of the -- none of those 6 to 7 thousand tons of chrysotile was the cause or contributed to the development of any of these 17 mesotheliomas?

A. I would be reluctant to comment just because I really haven't read all the paper.

Q. Well, here's Dement's paper. You are familiar with this one.

A. Yes.

Q. This is the other plant, correct?

A. Yes.

Q. And in that plant they had less than 2,000 pounds total of crocidolite was ever processed in that plant compared to 6

to 8 million pounds of chrysotile annually over a period from the '50s to 1975, so that's 20 times -- that's 120 million pounds against 2,000. And they found in their subsequent mortality study that there was a case of mesothelioma in that group who worked in that plant for 35 years.

It's your opinion, is it not, based on what you've told this court earlier, that none of that 6 million or 120 million pounds of chrysotile had anything to do with that individual's mesothelioma. Is that accurate?

A. Well, again, if you look at the totality of what Dement published, there was fiber burden analysis done in these studies.

Q. There was no fiber burden analysis done in this mesothelioma case, was there?

A. Of the one mesothelioma case?

Q. Correct.

A. I don't know.

Q. Okay. Well, is it your testimony that everybody in the plant had crocidolite in their lungs because they used 2,000 pounds of it totally compared to the 120 million pounds of chrysotile?

A. I don't think you have any way of knowing that. But I think what you can do is look at the fiber burden tissue analysis that I mentioned earlier to see whether or not there

is evidence that there was above background levels of amphibole in that.

Q. And you said we shouldn't really be using fiber burdens for causation, correct?

A. Well, I said that it wasn't the sole determinative of causal associations. I think that you can use fiber burden analysis to determine whether or not there was amphibole exposure or not.

Q. Now, one thing about this study, even though they only found one, the only -- the definition of who was the cohort started in January of 1940 and the cutoff date was 1975. You would agree that 35 years is just the beginning -- or maybe the mid point of the latency period for mesothelioma.

A. Yeah, I think that's about the mid point, yes.

Q. So this cohort really needs further follow-up to see if, in fact, there is going to be more than one mesothelioma in that group. You would agree with that.

A. I think so.

Q. Okay. And the follow-up was done with the fifth through eighth revisions of the International Lists of Diseases and Causes of Death classification, so that death certificates were coded.

So there's an issue that there may have been other mesotheliomas in the case in the plant that just were not coded properly.

A. I can't comment. I just don't know.
Q. Okay. Now, you know that there -- I think you said on direct that the only people that's ever been reported -- maybe I'm being too general about this, that got mesothelioma from chrysotile exposure were minors miners in Canada and in Italy.
A. Yes.
Q. You are aware that there have been studies, peer reviewed published studies -- here's one from Germany -- where the German Federation State of Saxony had records of 843 asbestos induced mesotheliomas, and they were able to determine a considerable percentage of those mesotheliomas were solely due to exposure to chrysotile asbestos. You're familiar with this article, correct?
A. I'm familiar with the study. The only major problem with it is really there was no occupational exposure history which was reliable in these cases, and so you really don't know what these individuals were exposed to.
Q. Well, we know that all the asbestos-based products were made from raw asbestos that was primarily imported from the former Soviet Union. The Soviet Union had one of the largest chrysotile mines in the world, correct?
A. Right, but jumping to the fact that these individuals were not exposed therefore to amphiboles is very difficult. So the exposure types that happened in the Soviet Union and Eastern Europe at that time, I think it would be safe to say

that we really don't know what the exposures were.
Q. Well, we know that the chrysotile came from the Soviet Union and it came from Canada and it was used to manufacture asbestos cement pressure pipes free of amphibole asbestos, according to the authors, correct?
A. That's what it says.
Q. We also know that the authors must have had some occupational information because they were able to categorize people as having amphibole-only exposure, amphibole and chrysotile exposure, chrysotile with possible amphibole exposure, and chrysotile alone exposure.
Now, you don't believe that these authors who published this paper in a peer reviewed journal would just make those numbers up, do you?
A. I'm not saying they made it up. All I'm saying is it's unclear about how they made that determination. It's just not clear.
Q. But their determination was that out of those 843 cases, 67 of them were due solely to exposure to chrysotile with no amphiboles.
A. Again, how they knew that that fiber type was only chrysotile is unclear in the paper.
Q. They're not talking about miners.
A. No.
Q. They're talking about end product users or people that

manufactured products, correct?
A. Yes.
Q. So that was the Carolina cohort. Now, we talked about Quebec. Let's talk very quickly about Balangero.
Now, I understand from Dr. Garabrant that somebody -- now, were you the one who uncovered Dr. Gunter's chapter from 2007?
A. I've read it. I don't know that I discovered it, but I read it.
Q. Where did you get it from?
A. I actually had it in another instance I was looking at non-asbestiform minerals exposure and saw it in that context.
Q. So you were just thumbing through a geology textbook --
A. Well --
Q. -- in a 50-page article and you ran across one sentence that talked about the fact that some tailings somewhere in Italy may have had 10 percent tremolite.
A. No, I was actually doing some research on non-asbestiform minerals. And I have read things as long as 50 pages before. And so found that sentence which I thought was interesting.
Q. And so the WR Grace lawyer in the bankruptcy proceedings that you participated in didn't have any input in you finding that?
A. No.
Q. Now, let's talk about this. Now, you agree that that one

sentence has no citation to it.
A. It has no citation.
Q. You don't know where that dump is located.
A. I don't.
Q. You don't know what was dumped in there. You don't know where it came from.
A. No. I mean the statement that Mickey Gunter made stands on its own. I don't know what reference source he had.
Q. Having -- having investigated non-asbestiform rock formations, do you find it unusual that everybody who studied Balangero from Pira to Piolatto to all those authors, all those Italian authors that found this rock in the late '80s and tested it, they didn't find any amphiboles, correct?
A. No, they didn't. And I don't find it unusual. I think that a lot of times the authors are focused on whatever they are focused on and don't necessarily look for other things.
Q. Mirabelli in 2008, they tested the mine tailing. Their mine tailings --
MR. SCHACHTER: Objection, Your Honor. He's mistaken. There was only one test. It was in 1989. And they're referring to -- he can't make things up -- well, he's been doing it a lot already, I apologize.
MR. GEORGE: Excuse me.
MR. SCHACHTER: Withdraw the objection.
MR. GEORGE: I take offense at that, Your Honor. I

don't make up anything less than Mr. Schachter did on direct.
Q. Let me ask you this. This is an uncited statement from the Mirabelli paper, correct?
A. Yes.
Q. It says, "Asbestos and the mine tailings which are crushed serpentine rocks left over after fiber extraction and which still contain up to 1 percent chrysotile fibers by weight, were removed by trains and lories."
That's what they said, right?
A. That's what it says, but the Mirabelli paper is interesting in that in one of the tables, I think it's table 2, they found tremolite contaminant in talc, and whether or not that is important in saying that something is tremolite free or not is a real question.
Q. You're kind of getting to where I'm going.
A. Thank you. I was trying to help you along.
Q. These trains and lories, where were they taking the tailings?
A. Where were they taking it to? I have no idea.
Q. They're taking them to the railroad. Why? Because they use them for ballasts on the the railroads throughout Italy. Isn't that what they say?
A. No.
Q. Look. "The mine tailings from Balangero were used in several areas of Piedmont as a ballast for road and railroad

construction and for courtyard paving."

So there wouldn't be some big mound of tailings at Balangero, at least through most of the time because they were transporting them throughout Italy.

A. I don't know the answer to that. I'm not sure.

Q. Now, these authors were aware of the tailings issue, correct?

A. Yes.

Q. This is not something that surprised them, oh, tailings. They say, We looked for people who were asbestos or tailings from the Balangero mine. They counted people who were living with an employee of the mine and individuals that were reported exposure to the mine tailings. And the reason why they wanted these people is because they didn't find any amphibole asbestos in those rock places.

Three cases were never employed at the Balangero mining site but had worked with asbestos, asbestos ore and mine tailings. Five cases occurred among persons exposed to mine tailings, three women who lived along with them.

In their chart they tell you -- now, here's where the tremolite comes in. There was a mill that treated tremolite contaminated talc from local mines. So there's local mines around where this Balangero mine is that has talc that has tremolite in it. At least according to these authors, correct.

A. Yes.
Q. And they say that not once but twice.
You don't know if it's those tailings are the ones that Mickey Gunter was referencing in his book, do you?
A. I have no way of knowing that.
Q. They say, Cases 23 through 27 neither worked at the mine nor reported circumstances responding to our definitions of environmental or household exposure. However, they were exposed to Balangero mine tailings used as a ballast. Possible exposure to tremolite may have also played a role in cases 23 and 27. Ballasts from the Balangero was used for the beds of railway lines up to the mid 1970s.
A. Right.
Q. And then it was later replaced by serpentines, some of which were contaminated with tremolite. That's the tailings that were contaminated with tremolite.
A. Right. But I think -- and I don't know if you are making this point or not, that the tailings had nothing to do with the mine itself.
Q. They are the refuse when you go to look for -- when you're looking for chrysotile, which is what the mine was about, you run into rock structures that have contaminants in it. And if they are visual contaminants, you don't go there. You blow that stuff up and you take it out. That's the refuse of your mining process.

A. Right, but I don't think anyone would say that all of those tremolite contaminated parts of the rock are removed. I don't see how that makes logical sense.
Q. Nobody in the world literature other than Mickey Gunter in one uncited sentence in a 50-page chapter has ever found amphibole asbestos in the Balangero mine, correct?
A. Well, I think that there's the Gunter chapter. And I also wonder about this issue of cattle and I notice that a co-author of Mirabelli, Fornero, mentioned that there was amphibole found in the surrounding areas from the cattle.
Q. Not anything that he's published in the peer reviewed literature, correct?
A. Fornero?
Q. Yeah.
A. Yeah, it's published.
Q. Well, we don't know if those cattle were hanging around by the talc mines, do we?
A. Well, I think that one doesn't have to make a big leap of faith to say that if there was amphiboles in farm animals who were not occupationally exposed, some of these individuals may have been exposed to amphiboles as well.
Q. It seems to me that whenever you are confronted with a situation where you have an extremely large percentage of chrysotile, you believe it's your job to try and find some evidence that there's amphibole. Is that what you -- is that

how you approach the question?
A. No.
Q. Okay. That changes their -- that updates from the Piolatto 1990 from 2 to 27. Now, they are not all miners. But even if we stuck to miners, it would go from two to six, correct?
A. Yes.
Q. And then the last one there is the Connecticut friction product plant which today Hodgson and Darnton still says that there was no mesotheliomas in that plant, right? You know that's not correct.
A. Well, and McDonald says that as well.
Q. Well, what Hodgson and Darnton is relying on is this study, and it was a plant -- it was a Raybestos-Manhattan plant in Connecticut. They did it by death certificates. And they didn't find any mesotheliomas.
Mary Jane Teta, on the other hand -- and that was a grant from the Quebec Asbestos Mining Association.
Mary Jane Teta, on the other hand, in her graduate days at Yale, went to the actual Connecticut tumor registry, right?
A. Yes.
Q. And what did they find in the tumor registry? Well, they found three cases. They had a female clerical worker who worked there for 30 years. They had another male possibly. And then a third case probable mesothelioma.

And in fact, since that time it's been reported that there are anywhere from six to seven, you agree that -- I think in your opinion there was five cases of mesothelioma out of that plant.
A. Yes.
Q. That's what you testified to, correct?
A. Yes.
Q. So that would change the ratio if we went to from zero to five, correct? So that means that three out of the four studies that they relied on to get this 500 to 100 to 1 ratio have been updated and have significantly more mesothelioma cases in them than when they did the original assessment, correct?
A. That wasn't the opinion that Hodgson and Darnton made in their letter. They thought that it didn't change their quantitative analysis at all.
Q. They don't know about what's in Connecticut, do they?
A. Well, I think what the question then becomes is are those cases in Connecticut attributable to work in that plant or is it attributable to different work? And that's -- that's a matter of some debate.
Q. But they're the ones to come up with this ratio. They're the ones who chose that cohort, correct?
A. They chose that cohort. The question, though, is for so-called new mesothelioma cases, are those mesothelioma cases

attributable to that work and that cohort or to somewhere else, and that's an important distinction.
Q. Well, when they commented on the fact that they're off by a risk of ten-fold, they were only commenting on the update of the Carolina cohort. They didn't mention the update of the Balangero cohort or the Connecticut cohort, correct?
A. Well, the only two cohorts that they mentioned in their update statement was regarding Connecticut and New Orleans. And they said that there was no quantitative change in their analysis.
Q. And where did they do that?
A. I don't have the letter with me. I could look at it.
Q. Not the letter that I just showed. The letter I just showed just said thank you, Dr. Loomis, for giving them new information about Carolina. It doesn't say anything about any other cohort, does it?
A. I think it does. I just don't have the letter.
Q. All right. Well, let's go to it, then.
There it is. Welcome, the new appearance. That's it. Risk derived.
A. Well, that's not --
MR. SCHACHTER: That's not the whole letter.
Q. That is the whole letter. The whole letter is only one page.
A. Well, I understand that, but you put two sentences up --

Q. I'll be happy to give it to you. You tell me when you read this letter if there is any other cohort discussed.
A. Let me read it and I'll tell you.
MR. GEORGE: May I approach, Your Honor?
THE COURT: Yes.
MR. GEORGE: In fact, Your Honor, I'll give you a copy of it as well if you'd like.
You don't want it?
THE COURT: No.
MR. GEORGE: That gives me an insight into your interest in this line of query so --
THE COURT: I'll wait to hear what he says.
(The document was tendered to the witness.)
Q. Dr. Weill, it shouldn't take you very long to read since this is only --
A. Well, you handed it to me about five seconds ago, so if I can just have one more minute --
Q. -- six paragraphs.
A. One more minute, I would appreciate it.
Q. Sure. That will give me enough time to click back to where I was.
A. Would you like me to read?
Q. You don't have to read it out loud, but you would agree with me --
A. No, it actually -- I would like to read it out loud.

THE COURT: Read the part that you want.

THE WITNESS: Yes, sir. "The absence of mesothelioma deaths in the New Orleans and Connecticut cohorts is statistically consistent with the risk of 0.01, although obviously more consistent with the mine's estimate of 0.001."

Q. Okay. Where does that talk about the update of any other cohort?

A. It doesn't.

Q. Okay.

A. It just says that their analysis is unchanged for the New Orleans --

Q. From the update of that single cohort.

A. No, from the original paper.

Q. My question to you is when they said that they were off by a factor of ten, they were only referring to the update of the Loomis cohort, correct?

A. That's correct.

Q. They didn't have any data, they didn't even recognize the fact that originally they used zero for the Connecticut plant and there may be as many as five or six. They didn't recognize the fact that they had two for the Italian mine when there may be as many as 27, correct?

A. Right.

Q. Okay.

A. And I'm just making the point that they didn't -- it's

not like they took the entire analysis and threw it away because of the information that they thought they knew about the North Carolina cohort.

Q. But it's not proper, is it, to say that the ratio is 500 to 100 to 1 when you know that the four studies that they based their entire ratio on have all been updated and all have significantly more mesotheliomas than the original estimate.

A. I wouldn't accept the last part of that because there's a question of attribution. I would also point out again that their update was based on the North Carolina cohort and not on the New Orleans and Connecticut ones.

Q. Want to talk real quick, continuing on this potency theme. You're familiar with the recent paper from the British Journal of Cancer entitled "Estimating the asbestos-related lung cancer burden from mesothelioma mortality"?

A. The first author, please?

Q. It is McCormack, Peto.

A. Yes.

Q. Peto is the one we were talking about before.

A. Yes.

Q. And what they did is they looked at a cohort, correct?

A. Yeah. I should say I haven't had time to review this paper in detail, so I'd rather not comment on this one.

Q. But would you agree with me that their cohort numbers are different that Hodgson and Darnton?

A. I'll accept that, but I just haven't looked at the methodology in any kind of detail.
Q. But they are magnitudes different from what Hodgson and Darnton said, correct?
A. Again, I just haven't looked at the study in any detail. I know the study you're referring to, I just haven't looked at it.
Q. Let's look at dose response really quick. This is what the regulatory model is. And what the regulatory model assumes is there is no threshold. And if you assume there is no threshold because it hasn't been demonstrated, that means every exposure is going to entail some risk, correct?
A. That's what that kind of model assumes, yes.
Q. And the more exposure you have, the more your risk is.
A. That's right.
Q. That part -- part of the epidemiology in asbestos has confirmed that at least some part of the dose response curve is linear. We know the high levels of exposure, that there is a dose response relationship that is linear.
A. I wouldn't say it that way.
Q. Okay. Now, what you're trying to postulate and what your view is that it's not linear, it's this S-curve, and that there is some low dose threshold which you can't quantify and science can't quantify below which there is no disease, correct?

A. It can't quantify the precise level of it. I think we have demonstrated that there is a threshold.
Q. Well, now, you talked about Berman and Crump in your direct, correct?
A. Yes.
Q. And you're aware of this because we talked about it in October --
A. Yes.
Q. -- that Berman and Crump, actually, instead of the three dose response curves that you showed the court that all showed no risk, they actually see an increased risk at lower doses. It's a supralinear curve. It's that A, correct?
A. I'm not sure that's what they were saying.
Q. Let's see.
A. I think what they were doing was postulating that any of these curves are possible.
Q. Well, let's see what they said. They say access to raw data from the cohort exposed to crocidolite in the mines and mills at Wittenoom, the three sub cohorts exposed to chrysotile in the mines and mills in Quebec, Canada, and the cohort exposed primarily to chrysotile at the textile plant in Charleston allowed us to formally test the linearity assumption in these cohorts. A supralinear exposure response was found in all five cohorts.
Meaning, they found curve A in all five cohorts that

there was more disease at lower levels than there were at higher levels, correct. Proportionately.

A. The issue is, though, that the exposure levels they're talking about wouldn't be characterized as low levels.

Q. Well, they say at the same time, If the true relationship for mesothelioma is supralinear, this would mean that risks at low exposure are larger than what would be predicted by the linear model.

They're saying that this regulatory scheme of a linear dose response model may be underestimating the risk that people have at low doses, correct?

A. The only issue I have with this part of the paper is I just don't know what they mean by low exposures because they didn't define it.

Q. This is a paper that you've read and a paper you rely on.

A. Yes. Yes.

Q. Let's talk about the federal register. And this is all about the risk analysis, and I think you told us about a risk analysis from early 1980s. You know they redid it in 2008.

A. Yes.

Q. And when they redid it, although OSHA stated in the preamble of its 1994 final rule that there is a remaining significant risk of material impairment of health or functional capacity at the 0.1 fiber CC limit, OSHA concluded that this concentration is the practical lower limit of

feasibility for measuring asbestos levels reliably. And the Mine Safely Health Administration agrees.

Just like we talked about, that as low as they can go is .1 and still be able to regulate exposure.

A. There may be a feasibility issue about regulating at those levels.

Q. So in 2008 a search of the peer reviewed scientific literature yielded many new articles that continue to demonstrate and support findings of asbestos induced lung cancer, mesotheliomas, and asbestosis, consistent with the conclusions of OSHA and the Agency for Toxic Substances Disease Registry, ASTDR (sic). Thus, in the scientific community, there is compelling evidence of the adverse health effects of asbestos exposure.

Where did they find it? It's found at the mines. Asbestos exposure of miners can come from either naturally occurring asbestos in the ore or host rock or from asbestos contained in manufactured products. And they call those asbestos-containing materials or ACM.

That's what they wrote, correct?

A. Yes.

Q. Asbestos in manufactured products, such as electrical insulation, joint and packing compounds, automotive clutch and brake linings, and fireproof protective clothing and welding blankets, could present a hazard during activities at the mine

site that may cause a release of fibers. The presence of asbestos at the mine indicates that there is a potential for exposure.

And there's also a potential of exposure if you manipulate a product that contains asbestos, correct?

A. I would agree.

Q. MSHA has determined that OSHA's 1986 asbestos risk assessment is applicable to asbestos exposures in mining. In developing this final rule, the Mine Safety Health Administration also evaluated studies published since OSHA completed its 1986 risk assessment, and studies that specifically focused on asbestos exposure of miners. These additional studies corroborate OSHA's conclusions in its risk assessment.

And they go on to talk about cancer mortality, correct?

A. Yes.

Q. They say in its 1986 risk assessment, OSHA estimated cancer mortality for workers exposed to asbestos at various cumulative exposures (i.e., combining exposure concentration and duration of exposure). The Mine Safety Health Administration has reproduced this data in table IV-1 which shows that the estimated mortality from asbestos-related cancer decreases significantly by lowering exposure. This is true regardless of the type of cancer: Lung pleural, peritoneal, mesotheliomas or gastrointestinal cancer.

And you agree with that: The lower you go, the less likely it is you're going to get the disease, correct?
A. In general.
Q. And here's your tables. Now, at 0.1 fibers per CC, they're still estimating -- if you only did it for one year, they're estimating that there's going to be 6.9 mesotheliomas per hundred thousand exposed people. If you did it for 20 years, it would be 73. That's their risk estimate, correct?
A. And again, I don't know how they determined that so I'd be reluctant to comment on how they did that.
Q. But you would agree that this organization which is charged with mine safety, hence their name, they would do within their best powers to review the literature as they said they did to come up with this calculation.
A. Again, we've spent a lot of time talking about regulatory agencies and their role in making causal determinations versus health policy, and I can refer you to my earlier comments about that. I don't think that we can take whatever regulatory agency says and assign a causation argument to it.
Q. What they reviewed, unlike Hodgson and Darnton, is they reviewed Canadian miners, chrysotile; Italian miners, chrysotile; Indian miners, chrysotile; Brazilian miners; Canadian mines. They looked at all of the different literature that's out there. And they concluded that exposure to asbestos, a known human carcinogen, results in similar

disease endpoints regardless of the occupation that has been studied. That's different from your opinion, correct?
A. Yes.
Q. The term "asbestos" in the Mine Safety Health Administration' existing standards and this final rule is limited to the following six, one of which is chrysotile. Serpentine asbestos, white asbestos, right?
A. Yes.
Q. All right. Last thing. Encapsulation. And you talked a little bit about encapsulation. But you would agree with me -- and you know Bill Longo's paper.
A. Yes.
Q. He has pictures in there of what a new gasket looks like in a photomicrograph and what a gasket looks like that's been in a flange for a while. And you would agree with me that even in its pristine state, there are fibers that stick out of that encapsulated material.
A. I can't either disagree with that or not because I'm not an industrial hygienist. The only comment I would have is is that it seems over time that there may be an exposure of the fiber outside of the encapsulation. There are a lot of encapsulating materials, I understand, that degrade over time. I don't think that that's the only issue is whether or not the fiber is exposed or not. The question that I was mentioning in my direct was about its respirability.

Q. Well, and the respirability when all that encapsulating material, the matrix disappears because of the heat and pressure of where the gasket is and you're left with the residual asbestos fibers, using mechanical implementation to get that off a flange which generates dust is going to generate respirable fibers.
A. The question would be, then, how much of it is still encapsulated? Is there a little bit on it, a lot on it? And that's the question I can't answer.
Q. Right. That's not your area of expertise.
A. No.
Q. But you have seen and have heard from pipefitters in your asbestos cases that there are occasions when they tear apart those flanges where there is residuum of asbestos on both sides of the flange that has to be removed, correct?
A. Yes.
Q. And you just don't have the expertise to comment on what levels of dust are generated from what's happening. You leave that to the three industrial hygienists that already showed up.
A. Yes.

MR. GEORGE: Thank you very much.

THE COURT: Mr. Guy.

CROSS EXAMINATION

BY MR. GUY:

Q. Dr. Weill, my name is Jonathan Guy. I represent the future claims representative, Joseph Grier, sitting here in the courtroom. And we've been listening with great interest over the last four days to the science testimony.

Now, it's your opinion that you can only get mesothelioma from a heavy exposure of chrysotile asbestos, correct?

A. Yes.

Q. And that's not a new opinion, is it, sir?

A. No.

Q. And in fact, you've held that opinion for a number of years.

A. Yes.

Q. Did your father hold the same opinion?

A. I think that his opinion was similar. I don't know the exact nuances of all of it, but I think it was similar. I think that high doses of chrysotile that were known to be contaminated in his mind elevated the risk, but I think it's safer to ask him rather than me.

Q. And you started testifying in asbestos litigation in 2002?

A. Yes.

Q. And your opinion on these issues, which hasn't changed, correct, since 2002?

A. No.

Q. Your opinion is known to the plaintiffs where you've

testified.
A. Yes.
Q. And it's also known to the various defendants where you've testified.
A. Yes.
Q. You haven't testified at deposition or at trial on behalf of Garlock, correct?
A. No.
Q. But you did testify in the *Grace* case in 2008, correct?
A. Yes.
Q. And I can represent to you that Garlock was involved in the *Grace* case. They knew enough about your testimony and your opinion on these issues to engage you in this case, at least?
A. Who did?
Q. Garlock.
A. They asked me when the prospect of being retained first came up what my opinions were with regard to a lot of different medical issues so I'm not sure if they knew it before that conversation or after.
Q. When was the first time that you spoke to anyone who represented Garlock or Coltec or anybody involved with Garlock or Coltec?
A. I think it was in the fall of 2011, if I recall correctly.

Q. I think from your testimony this afternoon, you would agree that there is a debate in the academic circles about the issue of exposure to chrysotile and mesothelioma.
A. Yes.
Q. Do you have any reason to believe that Garlock wasn't aware of that debate in the 2005 to 2010 time frame?
A. I don't know what they were aware of or not aware of.
Q. But it's a fairly well-known debate in the academic circles concerning asbestos, correct?
A. During those time years? Yes.
MR. GUY: No further questions, Your Honor.
THE COURT: Thank you.
Mr. Schachter.

REDIRECT EXAMINATION

BY MR. SCHACHTER:
Q. Dr. Weill, our question here relates to specific products. And we've heard about this debate. And, of course, in any situation where we would be applying Federal Rules of Evidence under which the plaintiffs will have the obligation to prove by a preponderance of the evidence that their evidence on that debate is even admissible.
So I'd like to ask some questions about methodology. It is true that Dr. Lemen has written an article, correct?
A. Yes.
Q. And you have actually worked on that article in your

report; is that correct?
A. Yes.
Q. That article that he wrote started out, as explained either in your report before the court or in Dr. Garabrant's, started out as an affidavit written for Waters and Kraus that he had republished, correct?
MR. GEORGE: I'm going to object to the characterization for lack of foundation.
MR. SCHACHTER: It's in the Garabrant report under Rule 104. It's almost word for word the affidavit in 2001 when Mr. Smith-George was working on that case.
MR. GEORGE: Dr. Garabrant is not here and he's asking of another witness. I don't know if that witness --
THE COURT: Well, he can answer if he knows.
THE WITNESS: I only read that in the Garabrant report. I don't know the answer to that.
Q. Okay. But you did analyze the Lemen article in detail, right?
A. Yes.
Q. And when Mr. Smith-George asked you about it -- first let's get clear where it was published. It was published in the International Journal of Occupational Health, some title like that. And that's the same place that the Laura Welch article that's the brief that Mr. Smith-George projected and read from was published in.

A. Yes.

MR. GEORGE: It's George, by the way.

MR. SCHACHTER: I'm sorry, Mr. George read. I apologize.

THE COURT: It's a cool name, don't mess it up.

MR. SCHACHTER: I thought the first one was cool, Smith-George. I apologize.

Q. It was published in an article and the editor of that article currently is a Mr. Egilman who is a plaintiff's expert.

MR. GEORGE: Dr. Egilman.

Q. Dr. Egilman. It's late in the day.

A. That's my understanding.

Q. Right. Whose opinions have been excluded because he manipulates the evidence.

MR. GEORGE: I'm going to object to that characterization. Lack of foundation. It's argumentative.

MR. SCHACHTER: It's in our 104 documents and I withdraw that.

THE COURT: Sustain the objection.

Q. Sir, you looked at the Lemen article in detail and the criticism that you discussed here was that the article is looking at -- I guess he asked you about the brake article. And the brake article deals with something we've -- we've got plenty of evidence on. I mean, it's not like no one has

written in the literature that low dose products don't create disease. There are a host of case control studies on that, right?

A. That's right.

Q. There is not a statistically significant association if you look at all the studies, right?

A. Correct.

Q. All right. So then the issue on the law is can Dr. Lemen in his article marshal evidence under the Bradford Hill criteria without a statistically significant association?

A. No, that's what I -- the point I was trying to make.

Q. Okay. Would you explain that to us.

A. Yeah. The Bradford Hill criteria were -- or factors as some people call them, were meant to be applied only to associations that were already proven to be statistically significant. The statistical significance is the first hurdle.

The second hurdle, then, is applying the Bradford Hill criteria to that statistically significant association in order to make a causal association.

Q. If we represent that the Federal Judicial Center's Manual on Scientific Evidence says exactly that and case law says exactly that, do you find that to be a reasonable position in interpreting how science actually works?

A. It's very reasonable.

MR. GEORGE: Objection. Lack of foundation, Your Honor, for him to speculate on what the law is.

THE COURT: Overruled. Go ahead and answer.

A. It's very reasonable. And just a real life example. If we were to say that there's a statistically increased chance of getting hit by a bus on Tuesday, we wouldn't say that there's a causal association between Tuesday and the bus accident.

We would then have to apply Bradford Hill criteria to see if that statistical significant increase was then causally associated. Those are two separate processes.

Q. And if we hadn't found the association in the first place of statistical significance, we wouldn't have to go through the whole Bradford Hill stuff, right?

A. Nor should we.

Q. Nor should you.

Okay. So if an expert in this so-called debate is relying on articles that employ a methodology that science rejects and that the law rejects, that wouldn't be supportive, would it?

A. Not in my view.

Q. Now, in science Mr. Smith -- Mr. George read at length from the brief that was written apparently by some lawyers on behalf of Ms. Welch and 53 of the people that she got to sign it. You've looked at those signers, right?

MR. GEORGE: I'm going to object to the form of the question. There is no evidence that any lawyer wrote any part of that brief. No foundation for this expert to --

MR. SCHACHTER: Well, in her deposition that's what she said. I mean...

MR. FINCH: That mischaracterizes Dr. Welch's testimony.

MR. SCHACHTER: Apparently Dr. Welch herself wrote the brief to the Michigan Supreme Court.

Q. In any event, is it customary in scientific circles, when you go to scientific meetings, to stand up and read briefs and decide issues based on what's in briefs from lawyers -- from scientists who are writing their briefs to courts?

A. No.

Q. Okay. So in a debate, would that be something that if it were a scientific debate would even be allowed?

A. No.

Q. Now, Mr. George did some math with some of the studies and he talked about the Connecticut plant and that there are more cases that have come out or allegedly come out of a Connecticut plant. Are you aware of the authors of an article that have done that, that have brought that up?

A. There was an article by Murray Finkelstein and a lawyer, I believe, that outlines some of the issues surrounding the follow-up in that plant.

Q. Okay. So the cases he was talking about adding are cases in a lawyer's article --
A. Yes.
Q. -- that's gotten into the peer reviewed literature somehow?
A. Yes.
Q. All right. And are you aware that those cases were actually attributable -- that people were making claims for amosite or amosite product exposures that includes those claims?
A. Yeah, I'm aware of that.
Q. And that wasn't disclosed in those articles, right?
A. Right.
Q. Would that be evidence that would be allowed to be put on the scales in a scientific debate?
MR. GEORGE: Object to foundation, speculation. Lack of a standard.
Q. Yeah. Does it comply with the scientific standard -- I withdraw the question, Your Honor.
Q. Does it comply with the scientific standard to publish an article claiming exposure from a substance and not disclose all the exposures that the person has?
A. No, that wouldn't be scientifically valid.
Q. And lastly, we've heard about a lot of public -- various agency reports. And the one that I think that Mr. -- well,

this is the second to last issue.

Are you aware that the World Trade Organization that Mr. -- zoom in a little. Mr. George read to you from the panels' findings from the World Trade Organization. You're aware, are you not, that the World Trade Organization resolves disputes between companies on trade issues and that that panel was forced to decide whether France was using a proper risk assessment methodology, a government procedure, right?

A. Yes.

Q. And the panel, actually, were not medical people, right?

A. That's what I understand.

Q. In fact, the three people, one of them, his qualifications were he had an advanced degree in French literature, right?

A. I wasn't sure of that.

Q. Well, if we look it up on the internet, that's what it shows. But they were very careful to say they weren't weighing in on scientific issues, weren't they?

A. Yes, they were.

Q. And in fact, they said, "The panel feels bound to point out that it's not its function to settle scientific debates, not being composed of experts in the field of possible human health risks posed by asbestos. Consequently, the panel does not intend to set itself up as an arbiter of the opinions expressed by the scientific community." Was that what they

said?
A. That's what they said.
Q. And that makes sense. You're not asking those kinds of people.
If you're going to have a scientific debate, would you weigh what was said or written by somebody with an advanced degree in French literature?
A. No, without any disregard to French literature.
Q. Okay. Lastly, Mr. George read you an update from some mining agency, and I apologize if I misstated the agency. And in that they said they had looked back at the risk assessments from '86 at whatever time, and that their conclusion was that they were going to stay -- even with all the other science since, they were going to stay with the level of .1 fiber per CC as the standard, right?
A. Yes.
Q. Even that agency erring on the side of over protection felt that was good enough to protect their workers, right?
A. Right.
Q. And if the exposure from gaskets does not exceed that level and there's no epidemiology that products that released even more than gaskets creates disease, is there any reason we should be concluding here that a viable methodology exists that passes muster in scientific terms for concluding that gaskets or packing in real world, real people, not rats, not

miners, real gasket users, that it causes mesothelioma in those people?

A. Not in my view.

MR. SCHACHTER: Thank you, sir.

MR. GEORGE: Your Honor, can I just redirect on one issue?

THE COURT: Okay. Go ahead.

RECROSS EXAMINATION

BY MR. GEORGE:

Q. I just want to talk to you about Sir Bradford Hill. You would agree with me that Sir Bradford Hill himself in his speech which laid out these criteria, one of which is association, right?

A. Right.

Q. How much association there is. That's one of the nine. He said in his speech that -- hold on one second.

There are -- here, then, are nine different viewpoints. Those are his nine criteria. Not even criteria really, they're nine different viewpoints, right?

A. Factors, consideration, yeah.

Q. Biological plausibility, coherence, analogy, association.

And he says, From which we should study association before we cry causation. What I do not believe, and this has been suggested, is that we can usefully lay down some hard and fast rules of evidence that must be obeyed before we accept

cause and effect. None of my nine viewpoints can bring indisputable evidence for or against the cause and effect hypothesis and none can be required as a sine qua non. What they can do with greater or less strength is to help us make up our minds on the fundamental question: Is there any other way of explaining the set of facts before us? Is there any other answer equally or more likely than cause and effect?

And he goes on to say on tests of significance, these confidence intervals, these mathematical formulas. No formal tests of significance can answer those questions. Such tests can and should remind us of the effects that the play of chance can create and they will instruct us in the likely magnitude of those effects. Beyond that they contribute nothing to the truth of our hypothesis.

That's what Sir Bradford Hill had to say about statistical data, correct?

A. Yeah, and I think that's exactly my point. I think statistical significance alone doesn't prove causation, but in order to consider those factors, you must have that.

MR. GEORGE: Thank you.

Thank you, Your Honor.

THE COURT: Okay. Let's shut down for the day. We'll be back at 9:30 in the morning.

UNIDENTIFIED SPEAKER: Can we get their batting order for tomorrow?

THE COURT: Okay.

MR. CASSADA: Thank you, Your Honor.

Tomorrow we will call Professor Lester Brickman, Rick Magee, and if there is time we will call John Turlik.

We're chagrin to report that we are behind where we hoped to be at this point. We would be open to starting earlier tomorrow if -- at the court's pleasure. Of course, we'll be happy to start at 9:30.

We've agreed to turn the court over to Mr. Guy and his -- and one of his witnesses at 3:30.

THE COURT: Tomorrow afternoon?

MR. CASSADA: Yes, sir.

THE COURT: Let's just start at -- well, let's start at 9:00.

MR. CASSADA: Okay. Thank you.

MR. SWETT: Your Honor, there is some other housekeeping.

THE COURT: Okay.

MR. GUY: No, the only thing I would add to that, Your Honor, is so that we can expedite things, the parties have agreed that as to the experts on inflation and discount rates, that we don't need to go through the qualification process other than to satisfy the court that they are qualified. So we're hoping to do that much more quickly.

THE COURT: If y'all are satisfied, I'm satisfied.

MR. GUY: Thank you, Your Honor.

MR. SWETT: Good afternoon, Your Honor. There are a couple of matters having to do with scheduling that have affected importantly the activities of next week.

The first has to do with the debtors' desire to bring rebuttal to the committee and FCR's science presentation which will take place on Monday and Tuesday and possibly spill into Wednesday. The debtors propose to bring that rebuttal at the end of the three-week period. We have asked them to instead bring it when the committee and FCR science people leave the stand. In other words, when they -- when Mr. Finch and Mr. George and Mr. Frost have their last witness off the stand, the science rebuttal should come forward.

The reason for that is very practical. When we were heading into a two-week trial that had been scheduled as such for many months, Mr. Finch became committed to another trial, that of a living young mesothelioma victim whose case is going to trial in Charlottesville the week after this trial is to conclude with pretrial activities to take place during the final week of this case. Mr. George, likewise, made professional commitments for what is now our third week. And Mr. Frost made a personal vacation plan and cannot be here at the end of the three-week period.

So we're in something of a bind. I mention it now. I hope that we can work it out with the debtors but initial

efforts were unsuccessful in doing so. And barring an agreement, I'm going to have to apply to you to direct the debtors to bring their science rebuttal in the middle of next week instead of at the end of the three weeks.

That's the first point. I don't know if Mr. Cassada wishes to speak it.

MR. CASSADA: Your Honor, at this point we're working with a time period that Your Honor gave us. We're not even done with our case in chief science case. We're moving from science to social science tomorrow. And one of reasons is because of the limited time we've been allocated and the necessity that we choose carefully and prioritize witnesses to make sure that we get on -- get witnesses on in an order of priority. So we can't agree to let the committee dictate the order of our witnesses.

And we do have a plan for the witnesses we'll call. We've made those arrangements with the witnesses and we're ready to proceed on the current schedule.

And I might add, Your Honor, I gave up a vacation for the second week, so a lot of people have made sacrifices to be here and to try to conclude this trial within the time allotted by the court. But we are at peril now getting through our witnesses.

THE COURT: I don't think I can force them into any set order of witnesses. I'll ask y'all to try to accommodate

that if you can. But I think I've got to leave it to the parties to try the case the best they can in the order in which they can try it. So let me just ask you to work together as best you can and see where we go from there.

MR. SWETT: There is another matter, Your Honor, which, again, has the been the subject of some discussion, so far not conclusive between the parties, and it has to do with some witnesses that we put on our order of proof as rebuttal.

They are witnesses who were not named previously. It's my contention that they were not required to be named previously. They are -- will testify if at all only in rebuttal of the factual aspects of Mr. Turlik's testimony and Mr. Glaspy's testimony.

The reason I need them, or at least need to hold them in reserve is because another witness whom I had planned to have available to respond to Mr. Glaspy and Mr. Turlik as a fact witness, Mark Iola, is unable to be here at all. He is out of the country or otherwise unavailable for the entire three-week period.

And so when I learned that very close to the time of trial, I made the decision that I had to be able to put down these other two people at the appropriate time as rebuttal.

Now, this takes place in the context of a case management order that did not speak specifically to rebuttal witnesses. We were all required to make a preliminary list of

our fact witnesses last December and then to give a final version of the same list some time later. There was no specific requirement as to rebuttal. Both sides put in their witness disclosures reservations of rights to call whoever they needed for rebuttal or impeachment.

When we were apoaching the pretrial conference, I wrote a letter to Mr. Cassada and I said it has been our experience that rebuttal witnesses need not be named unless and until they are to be used. We had a discussion about that. Mr. Cassada said he disagreed.

We then had the pretrial conference. You may remember we discussed various aspects of the issues raised in my letter. The debtors chose not to speak, not to raise the issue of my notice to them that I did not feel obliged to name my rebuttal witnesses in advance of deciding that they had to be used.

When it came time to give the order of witnesses, the debtors put down rebuttal witnesses for the first time. Up until then at every listing they have said we reserve the right to call anybody in rebuttal without naming them.

Now, Coltec gave its order of witnesses on the same day, July 15th, as the debtors did. And they put in their order of witnesses, we reserve the right to call anybody without naming them for rebuttal or impeachment.

Our order of witnesses came due on July 22nd. And

in an abundance of caution, even though I do not believe it's fairly required in the circumstances, I listed Mr. McLain, Mr. Roderick Paul, and anyone else needed for rebuttal. In that last reservation of rights I was in line with Coltec.

Now, I got an email from Mr. Cassada the other night saying he notices these two names and he's going to object. And I hope that further discussion will come to an accommodation. I'm willing to do what I can to satisfy him that he's not being ambushed. They are, after all, rebuttal witnesses who will only testify if needed to respond to something Mr. Turlik or Mr. Glaspy say, and I don't know what they're going to say yet.

So that's the problem. And if Mr. Cassada wishes to speak to it now, I'll yield the podium. But barring an agreement, I'm going to need some relief from the court.

MR. CASSADA: I don't want to unnecessarily prolong this before Your Honor, but they can only call a rebuttal witness they hadn't named if something unanticipated had come up. Mr. Turlik and Mr. Glaspy submitted expert reports back in February and they had an opportunity to submit rebuttal reports, and in fact did do that in the form of two lawyers when the rebuttal reports were due in April.

Back in December while we still had a fact period of discovery left, I had noticed on their list of potential witnesses the possibility of them calling lawyers that were

unnamed on their side. They said they might call lawyers that we had named.

So I wrote Mr. Swett and I told him that we don't want to have the possibility of a plaintiff's lawyer appearing at trial to testify unless we've had an opportunity to depose them. And he assured me at that time that he had identified all the lawyers who might show up.

So it was certainly reasonable for us to believe that we had an opportunity to depose any witness he might call, particularly any witness he might call on issues that he's had fair notice of for a very long time.

And I might add, we don't intend to call any witness in rebuttal who has not been produced for deposition. We haven't -- I suppose there might be some surprise testimony. We might ask for leave of court to do that then. But we have not taken the position that the parties -- that either we or the committee can just leave unnamed witnesses they might call and claim or answer to some surprise rebuttal testimony.

So we do object to their bringing these witnesses out of thin air who we never had a chance to depose. We don't even know what they are going to testify about.

MR. SWETT: I don't either until I have heard Glaspy and Turlik. If it helps, I can try to arrange for these people to submit to deposition before they take the stand, but I need to the flexibility to have rebuttal.

THE COURT: I think that's fine. It doesn't sound to me like there is any kind of ambush or anything going on here. It just that this is a dynamic situation and things happen and things come up and it wouldn't be -- wouldn't be a trial if there wasn't a surprise or two.

But I will ask you to make them available for some sort of examination prior to trial, if only for my benefit that I think that would probably speed things up during the trial rather than to have somebody have to do it at the trial.

MR. SWETT: Yes, sir.

THE COURT: All right. Well, we'll see you at 9:00 tomorrow morning.

ALL COUNSEL: Thank you, Your Honor.

(Evening recess at 5:37 p.m.)

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UNITED STATES DISTRICT COURT

WESTERN DISTRICT OF NORTH CAROLINA

CERTIFICATE OF REPORTER

I certify that the foregoing transcript is a true and correct transcript from the record of proceedings in the above-entitled matter.

Dated this 26th day of July 2013.

s/Cheryl A. Nuccio
Cheryl A. Nuccio, RMR-CRR
Official Court Reporter